SUPPLEMENT

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(Continued.)

TRACHOMA.

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Bearing in mind the desire of the Executive Committee that at this Congress the preventive aspect of medical practice should be especially kept in view, trachoma has been selected as one most suitable subject for discussion, for no eye disease can show, as the result of neglect of ordinary rules of hygiene, such an increasing incidence of cases each one of which is threatened with interruption of education or employment according to the age of the infected person and with the possibility of a permanently reduced industrial efficiency or even blindness as the result of disabling sequelæ. On the other hand the institution of adequate treatment and the adoption of elementary principles of hygiene will lead to its ultimate disappearance from a district or country and with that a menace constantly present to adjacent communities.

In broad outline one has only to contrast the result of the preventive measures adopted in the British Army in France in the late war, where both the armies as a whole and the civil population were protected from the heavily infected labour companies that arrived early from Egypt and China, and the low incidence amongst the Australian troops in Egypt and Palestine, for information on which points I would refer you to the reports of Sir James Barrett, Colonel Eason and Major Mitchell, with the outbreak following the disbandment of the forces of the Napoleonic and Russio-Japanese Wars and its prevalence to-day in Kentucky and Southern Illinois. The Prussian and Belgian armies also witnessed the same diminution of cases as the result of strict measures of quarantine, notification and treatment which reduced the figures in the former from 32.3‰ in 1867 to 1‰ in 1912 and in the latter from 200‰ in 1840 to 0.67‰ in 1900. One can further add as an example of the benefit of the application of the same principles the comparison between the wholesale infection of the natives in Egypt up to 90% to 95% and in like proportion of the children of the primary school if that at Tanta can be taken as typical with their trachomatous 21.4% of the totally blind due to lack of control and enlightenment, and the reduction of the incidence in the schools of Jerusalem through supervision of the children from 31.3% in 1913 to 13% in 1916 in circumstances not noted for indigenous hygienic procedures.

In Western Australia taking 10,000 consecutive private patients 3.26% showed evidence of trachoma. Dividing them into an earlier series of 3,540 and a later, of 6,460, a division determined only by my system of card filing, the percentage in the former was 2.32 and in the latter 3.8. This would appear to be an increase, but on dissecting the cases into new and active on the one hand and old, quiescent or apparently healed on the other, the new and active in the earlier series formed 1.8% of all eye cases or 79.2% of the trachoma cases and in the second later series 1.7% and 46.0% respectively.

The diminution of active cases I attribute to an increase in the standard of living and education together with an easier access to treatment curtailing the infectivity of focal cases.

While the fact of its infectiousness is not disputed the preventive measures that ought to be established as the most appropriate and essential, rest to a large extent on empiricism, the actual causative organism and therefore its bionomics and the usual channels of its transference being as yet not scientifically established. While we leave on one side the contention that any chronic non-specific irritant can produce the disease in the presence of certain constitutional factors, it must be acknowledged that the influence of the latter requires research. Although I have not found any association between tuberculosis and trachoma as has been suggested by oculists in America, in my experience the most resistant cases and the most liable to relapses and pannus occur amongst patients of the scrofulous type with carbo-hydrate intolerance, in whom frequently an eczematous keratitis will complicate the corneal picture and possibly interfere with a concentration of the local reaction against trachomatous infection. Bäck found much the same. Doubtless the greater number of cases among the less well to do classes is to be attributed not only to the increased chances of infection and the fewer articles of toilet reserved for personal use, but also to the general lowering of resistance through some inadequacy in the food supply in quality, if not in quantity. For in Western Australia patients arrive from areas where there is no overcrowding and it is impossible to get away from unlimited supplies of fresh air by day and night, yet there is often in these districts a lack of such necessary articles of diet for the young and growing as fresh milk and fresh vegetables

which must result in a lowering of the general if not of the local resistance.

This leads me to remark that the majority of patients are infected in childhood and adolescence, fewer in maturity and very few in old age, if any. This was also Straub's view and that of Morax and Müller based on their experience in Egypt. In Palestine and Baku the same is found. In Western Australia 8.3% of the outpatient eye patients (new) at the Sick Children's Hospital, Perth, are trachomatous, but the percentage at the Perth Hospital, although I have not the actual figures, for a corresponding period is less and the patients sent there are generally of long standing. In a limited series of 3,540 private patients of all ages the percentage was 2.32 and of these eighty-two, 33.0% were under the age of eighteen years and one-third of these under puberty. As in the older patients the disease had been in existence for some years at least, the greater number had been infected before fifteen years of age. This suggests difference in the types of the disease or some other undetermined modifying condition to reconcile it with the experience of H. V. Würdemann in the United States of America who writes that trachoma "is not a disease of children. It is seldom seen before puberty even in families in which the adults are affected."

Then the unilateral cases of trachoma present a problem of local immunity difficult to explain, although no more so than the inability of Germaix and Bach to inoculate their own healthy eyes either by rubbing in or by injecting subconjunctivally emulsified trachomatous material. It has been said that the second eye in these patients ultimately becomes trachomatous, but that is not my experience. I have had in my practice cases which have remained unilateral during over thirty years of observation by different oculists.

These apparent exceptions at present so baffling await discovery of the infecting agent. Meanwhile certain experiments seem to establish some definite limits to the range of the activity of the virus. Nicolle, Cuenod and Blanc from their experiments conclude that a fly in contact with an infected eye or linen contaminated within six hours can transmit the infection for twenty-four hours at least and in this time a fly can travel a considerable distance. This virus, filtrable in their opinion, is destroyed by heating to 50° C. for half an hour, while Kooy found her cultures resisted drying at 32° for half an hour and in ascitic fluid 45° for one hour killing being secured at 50° C.. Botteri found loss of infectivity after three-quarters of an hour at a moist temperature of 0° to 10° C. or after four hours at 15° C..

The probable length of the incubation period has also experimental data bearing on it. Hess and Römer failing with a filtrate of an emulsion to produce any lesion in apes, found with the unfiltered material after three to four days of inflammatory reaction an evident infiltration in eight days; Bajardi after grafting trachomatous tissue into the conjunctival pouch or using a subconjunctival injection of an emulsion saw a follicle formation in two weeks. Nicolle, Cuenod and Blanc using rabbits found the period to be nine to fourteen days. Addario and Greeff using blind subjects found in man a progressive infiltration about the eighth day.

Beyond this the question of the actual organism becomes contentious. The more common pathogenic organisms having been excluded, bacteriological discussions are at present practically narrowed down to the pathogenicity of the so-called inclusion bodies first described by Halberstädter and Prowazek in 1907 and the initial and elementary bodies of Lindner. Those have been described on the one hand as products of cell degeneration by such as Stanculeanu, Kiribucki and Bertanelli, but more prolonged acquaintance with them has tended to their recognition as organisms. Similar bodies have been described in swine fever, *epitheliosis desquamativa,* and spring catarrh, but the care required in the examination of the microscopic slides to identify the bodies is shown by Greeff's statement that he found some of the so-called occurrences were not of trachoma bodies at all and not all the so-called epithelial cells were even epithelium. He could himself distinguish on morphological lines such bodies as those claimed to be found by Blehar in the infectious vaginal discharge of children and by Hofer in scarlatina.

Lindner in the same way distingiushes the true bodies from those occurring in swine fever, spring catarrh, and *epitheliosis desquamativa,* a distinction supported by inoculation experiments which are positive with the former and negative with the latter. The most serious obstacle to their being regarded as typical of trachoma came from Heymann's discovery of bodies indistinguishable from the trachoma bodies in ten cases of *gonoblenorrhoea neonatorum* which Pfeiffer thought might be pathogenic. Herzog's view that they were a mutation of the gonococcus was not accepted. Halbertstädter and Prowazek recognized the full morphological similarity, but not the identity of the two bodies and traced the infection of a child showing the bodies back to the vaginal epithelium of the mother.

Lindner going further found them in the male urethra and in accepting the identity of the two bodies practically gave as his opinion that the non-gonococcal *blenorrhoea neonatorum* was a form of trachoma and that there was a trachoma of the genital passages. Noguchi and Cohen restrict the affection produced by the inclusions to the benign conjunctivitis the so-called "inclusion conjunctivitis" inasmuch as inoculation experiments on the higher monkeys with secretion from these lesions never produce trachoma, but only the same type of benign conjunctivitis and thus the occurrence in trachoma of these bodies would have to be regarded as a coincidence only. But Lindner distinctly states that the inoculation of monkeys from such cases produced an "infection quite similar to the one in trachoma." It must be remembered that Römer and Hess using whole trachomatous secretion found that, while the resulting lesion was microscopically trachoma, the clinical course was milder and contractions less than in man. On the other hand, the cases in the Fuchs clinic of inclusion blenorrhœa were to Lindner "clinically genuine trachoma" with some scar formation and Wolfrun quoted by Lindner after inoculating two men with inclusion blenorrhœa saw "genuine trachoma followed with the production of scar tissue." To me it appears, therefore, from the evidence available, often unfortunately in the form of abbreviated reports, that Noguchi and Cohen have not altogether repudiated Lindner's claims.

It may be well to remind you of Lindner's description

of the bodies. "In well fixed and stained (Giemsa) sections inclusion appears as follows: Blue, sharply outlined, round cocci-like bodies are seen in the cavities of the protoplasm of the epithelial cells. They multiply by special division and later on are found only near the wall of the cavity at a time when the red points appear in the cavities. These blue bodies which I called 'initial bodies,' are found not only in the cells but extracellular as well, sometimes even in great numbers. These extracellular bodies are very characteristic and can be easily recognized. Prowazek's inclusions are nearly always accompanied by these free 'initial bodies.'"

In smears "one finds dark blue granular inclusions in the protoplasm of the epithelial cells. At first small round or oval they apparently grow, become less dark and then fine red points appear in the masses. The red points increase rapidly in number and the blue masses gradually disappear."

Miss Kooy is so persuaded of the causative relationship of the inclusion to trachoma that she considers it possible in "nearly all cases sixty-nine out of seventy to make the diagnosis from a slide of conjunctival secretion and epithelial cells by demonstrating the different forms of the polymorphous micro-organism." She succeeded five times in growing these facultative anaerobes in culture when a sequence of formation of the different forms could be traced. Correlating it with the clinical picture the Prowazek bodies are probably the most contagious, being most numerous in the acute forms and yet disappearing most quickly under medical treatment and expression, whereas the elementary bodies are the most resistant and more numerous in the older cases, so that trachoma in the cicatrical stage could still be dangerous.

The topographical method of investigation of conjunctival infections has to Lindner's mind established the occurrence of a genuine acute trachoma, the bodies, both intra-cellular and extra-cellular being found distributed both on the bulbar and the palpebral conjunctivitis to the exclusion of other pathogenic organisms resembling in the early stages an epitheliosis of the superficial cells, although later on they may be found in nests in the depths of the epithelium and so escape detection in the slide from the superficial epithelial scrapings.

This advance raises the hope that it may be possible with experience to dispose of the doubtful cases, when an incorrect diagnosis may be productive of hardship, as for example among the immigrants into the United States of America and in the States where trachoma is notifiable and the trachomatous placed under surveillance. It may also be possible to further the development of the treatment attempted by Nicolle, Cuenod and Blaizot who by intravenous or subconjunctival injections of a virus prepared from the patients themselves secured a considerable improvement in the cases.

In the absence, however, of any vaccine the treatment of the condition must proceed along such lines as the pathology would suggest. Beginning as an epitheliosis the follicle formation is early noted and in time an extensive subepithelial infiltration appears affecting both the palpebral and the bulbar portions of the conjunctiva. Although the follicle is such a prominent feature, it is doubtful if it is the most important or characteristic, since the follicle can be produced by various irritants, but not the subepithelial infiltrations which according to Hiwatari occur, excluding tuberculosis and syphilis, only in trachoma. Hence, the absence of the mention of the follicle from Treacher Collins's definition of trachoma as "a contagious disease which is confined to the conjunctiva and which is characterized by a new formation in it of lymphoid tissue." In Hiwatari's view in the presence of the virus the appearance of the follicles in one part and not in another of the conjunctival covering of the lids and bulb is conditioned by predisposing anatomical arrangements, for example the wealth of cells in the subconjunctival layer and the nature and thickness of the epithelium. He does not agree on histological grounds with the description of the *substantia propria* as an adenoid layer, so that here one really has to deal with a widespread pathological process and reaction which in its origin contains a suggestion of a means of cure for the researches of Hiwatari demonstrate that in the cellular reaction the fibroblasts are among the first to proliferate not appearing merely as the follicles degenerate, but as a part of the tissue response. The breaking down of the granule is therefore on this interpretation as much a consequence as a cause of the cicatrization which may occur independently of the granules wherever trachomatous infiltration is present, whether "in the conjunctiva of the tarsus in the fornix, on the globe or on the cornea"; he quotes in support the demonstration by Ichikawa of the "cicatrization in the bulbar conjunctiva."

It would appear logical, therefore, that in the measures adopted one should bear in mind both the granules and the infiltration. The superficial applications of such as "Argyrol" can only diminish the superficial hyperæmia, wash off superficially lying bodies and help to cleanse the folds of any accompanying mixed infection, whereas the stronger applications, such as mercuric oxy-cyanide accompanied by massage or expression will by the expulsion of the follicles or the mechanical removal of papillary overgrowth facilitate the action of the drugs, traumatize the subepithelial tissue and produce as well a beneficial hyperæmia that will tend to accelerate the fibroblastic proliferation. Carbon dioxide snow applications affect both formations not only causing a necrosis of the cells of the granules but also, as Akiya has recently demonstrated microscopically, a necrosis in the subepithelial tissues as well. Mitchell claims from its use a cure in 20.0% of patients. The results from it in my own experience have been moderately good, especially in cases partially cured that tend to relapse in spite of the usual treatment and in those cases that seen late in which islands of trachomatous tissue are left in the interstices of coarse cicatrices. The intervals between the applications also add to the convenience of patients, but it is possible by too frequent or too prolonged applications to produce a undue amount cicatrization which is the main disadvantage to my mind to its use. In this it compares unfavourably with the more uniform and more pliable scarring of copper sulphate which has been in use since the days of the early Egyptians at least. In spite of the praise of the snow treatment by oculists of large experience I do not think its results are so preeminently better as to replace this well tried remedy. In my own work

after the mechanical removal of the follicle and hypertrophic growth and reducing any discharge I, as a rule, have recourse to solid copper sulphate, which in my opinion gives the best results of any method of chemical treatment in an average case, but unfortunately it fails to cure a pannus which outlasts a palpebral affection.

Of other non-surgical methods of treatment, such as radium and Sculco's newer treatment with *Nepeta citriodora* I have no personal experience and the reports are somewhat conflicting. But the method of Schiele adopted by Rudas, of Buda-Pesth, which makes use of iodic acid, seems to demand a trial as against all other methods. He claims to obtain by it a reduction of the necessary period of treatment from 64.47 days to 44.12 days and in the frequency of complications (ulcer and pannus) from 3.25% to 0.85% and visual disturbances from 3.07% to 0.51%.

It would appear logical to advocate the combined Kuhnt-Heisrath excision but, partly it may be from a fear of further contraction of the conjunctiva from the original trachoma or from a dislike to remove mucous membrane which may be saved, I hesitate to remove so much conjunctiva and indeed I find it but rarely necessary in the trachoma I experience in Western Australia.

The simple excision at an appropriate time, however, is extremely beneficial, especially if Hiwatari's free dissection be carried out. The combined excision is advocated in Egypt, in the Balkans and particularly by the Whites in America, but in Königsberg I believe it is not so much practised as formerly and I hope that the addition of carbon dioxide snow to the usual chemicals may help to diminish the number of cases that seem to require it.

All will welcome any treatment that will help to subdue pannus, this outpost of the subepithelial infiltration. Particularly worrying both to the physician and the patient are those cases in which the lid conditions apparently retrogress, but the pannus remains irritable and liable to relapse. Subconjunctival injections of mercuric oxycyanide in some cases do good both to it and the accompanying ulcers, but in others fail altogether and they are not the sovereign remedy some would have acclaimed it to be.

Peritomy too may be of benefit or a complete failure even when clearing away tissue down to the sclera. Possibly there in view of the pathology one's efforts are too localized and Hiwatari's method of subconjunctival dissection from fornix to limbus may be more rational and likely to be successful by the acceleration of universal cicatrization. This is possibly one of the reasons of the good results obtained by excision of the tarsus with free loosening of the conjunctiva up on to the bulbus. Undoubtedly the lightening and increased pliability of the lids after the excision of the tarsus promotes a healthier cornea, but I do not think the amelioration is dependent entirely on these and the removal of the mechanical factors that may be aggravating the pannus, because although they may irritate an already present diseased area, that condition is itself predetermined by a definitely established pathological process. However, the longer one practices, the more one inclines to the radical surgical operation of tarsectomy when it is evident that the other methods are failing or are too slow in securing an amelioration in that the vascularization of the pannus and the chronic hyperæmia of the semi-cicatrized lids are not steadily diminishing or when the ptosis is marked. Even in patients who do well, if they live in too distant an area for occasional examination or who have to return before the completion of the course of treatment, I begin to think the excision should be done, provided that the conjunctiva is in a fit condition to permit of the operation.

No matter what the method, it remains obvious to every oculist that treatment ought to be instituted as early as possible after diagnosis and particularly so in childhood.

As a rule I find the younger the child, the quicker the response and the less the undesirable sequelæ of gross cicatrization and corneal astigmatism. It is for this reason especially that I would raise the question of advisability of notification. It would emphasize the necessity of the recognition of trachoma in undergraduate days among medical students, impress parents with the possible harm that may happen to a child or any other person for that matter from any case no matter how harmless apparently in itself, and place teachers in a more authoritative position in excluding a child with suspicious eyes from school pending diagnosis. It has been notifiable in Glasgow since 1915. By 1916 eight States of United States of America had separate trachoma laws and twenty-three more classed it among the contagious diseases, so great a menace had it become.

At the French Colonial Congress in 1922 the disease was stigmatized "as a formidable scourge in our colonies and in certain centres of the metropolis" to combat which an anti-trachoma league was to be formed. So far as I know it has not yet formulated its policy, but from the context I should imagine it will not content itself only with therapeutics, but will seek to make its control a question of public health.

As a country waiting to be developed by immigration, it behoves us to take stock of the condition in Europe. Switzerland is free of indigenous trachoma. In Greece of 14,000 cases of eye diseases seen in six years 3,000 or 214.5‰ were trachoma.

In Italy (Northern) according to Basso, one hundred and ten per thousand in Liguria and one hundred and twenty-six per thousand in Lombardy (of the inhabitants) in Spain (Hirschberg and Menacho) in Seville 102.5 per thousand, Madrid fifty to eighty per thousand, in Bilbao sixty-four to 47 per thousand, according to Sydney Stephenson, in England six per thousand, in Scotland 9.3 per thousand and in Ireland 26.4 per thousand eye affections were trachoma.

I have not the necessary figures for Western Australia to permit of a complete comparison in terms of the population.

In America that draws immigrants from all quarters of Europe, for the years ending June 30, 1914, 11,966,897 aliens were examined and 22,984 had trachoma; as an indication of what numbers were already stopped at the ports of embarkation in the year ending June 30, 1906, 22,600 were prohibited at foreign ports from sailing.

It may be mentioned that in 1903, Congress had endorsed the action of the Secretary of State in declaring trachoma to be a dangerous disease, carriers of which

were not to be admitted into the States. Here we deal with much smaller numbers and persons are examined before exbarkation to guard against such dangers, but it is well to recall even in the face of efficient examination the concentration of the foci abroad, for I suppose that 10‰ of the cases seen at the Children's Hospital occur amongst families of immigrants from the Southern and Eastern portions of Europe.

Sir James Barrett after his experiences in Egypt apparently thinks that immigration restrictions such as prevail in America are too stringent and possibly it might be sufficient if admission were granted to trachomatous immigrants to enter here, to place them under some system of surveillance as that in practice formerly in Prussia and lately recalled by the Army Council Instruction on April 4, 1917, whereby a medical officer of health would be advised when such persons were settling in his area. Still it is a disease that ought to be eradicated here in two generations and until we can be sure that all immigrants and inhabitants, old and young alike, are fully seized of the importance of the hygienic measures involved and of the danger of communistic habits of the young in particular, from every point of view, one profitable method of preventing the occurrence of fresh cases must be the check on importation from without.

I myself have not found it necessary to isolate the trachomatous children at the Children's Hospital; in nine years I have known only two cases originating there in each of which the health of the conjunctiva was impaired one through injury, the other through phlyctenular attacks. It is only necessary to enjoin strictly rules of cleanliness and a rigid use of the individual towel and no common bathing water.

It has long been recognized that infection is more liable to spread from those that show secretion and hence the legacy of cases in a district after a wave of acute conjunctivitis has passed over it, although it is not in itself caused by any of the organisms known to produce such conjunctivitis. The infection may occur in the main through the common use of towels and handkerchiefs, washing utensils *et cetera*, but in our north-west whence several of our patients come and are dated from an epidemic of conjunctivitis among the aboriginals, such agencies do not explain the conveyance of it to the white population. Nicolle, Cuenod and Blanc's investigations which incriminate the fly, furnish more likely explanation and Netto's suggestion that the louse, the crushed up bodies of which produce a folliculosis resembling trachoma, may be an intermediate agent and may help to explain the spread in conditions of family overcrowding and insanitary ways of living. Although the detailed instructions to patients or guardians may require a little care the principle of the preventive measures is obvious.

The actual harvest of blindness varies on the whole with the opportunity and thoroughness of treatment. Thus, in Russia trachoma is estimated to cause 21.4‰ of all blindness and in the United States of America according to Loeb 9.4‰. In Egypt the percentage must closely resemble that of Russia, for 69.7‰ are ascribed to trachoma and acute conjunctival conditions and of that number 40‰ are due to the gonococcus on Meyerhof's estimation. Bishop Harman in London found only 0.21‰ of 1,855 blind school children blind from trachoma and out of 601 blind persons of all ages only 1.3‰. I can recall only two persons in Western Australia that qualified for the blind school and these were flagrant cases of neglect where the cornea had become staphylomatous. Yet it may be mentioned as an example of what can happen in spite of attention that Cuignet, the inventor of skiascopy, is said by Axenfeld to have lost an eye from this disease.

There are in practically all patients who have been left untreated until pannus has been well established, varying degrees of lowered visual acuteness which may blast the whole of a person's career, a burden that still too often an innocent sufferer has to carry because of parental ignorance or poverty. While I am not prepared to state the percentage of all cases of reduced visual acuteness due to trachoma, I feel I am safe in saying that at least 50‰ of those showing active trachoma with pannus approaching the pupillary area in adult life have vision reduced to at least six-eighteenths. If the pannus be confined to the upper fourth a reduction below six-ninths is unusual.

There is one special feature of reduced vision to be referred to and that is apparent myopia. One constantly comes across patients to whom either concave glasses have been given at some time and are no longer required and for whom only weaker glasses are required to obtain the same visual standard. It may be partly a question of the myopia diminishing due to a slight flattening of the cornea which had previously acquired too great a curvature from the softening of the pannus, but I think it is also in some degree due to an involuntary over-correction at the time of the original prescription, either because of a ciliary spasm produced by the attempt to increase vision dulled by nebulæ or because the apparent improvement in outline from the diminution of the images is mistaken for improved vision. I am, of course, referring to patients in whom accurate skiascopy on account of multiple nebulæ is well nigh impossible.

Whether the use of polarized light will assist at all in these cases remains to be seen. So long as we have to depend on subjective findings, I wish only to emphasize the liability to over-correction.

For later sequelæ, such as trichiasis, if an operation be required, a greater assurance of permanency of correction is in my experience obtained from a combination of the use of the Van Milligen mucous membrane graft with the Hotz-Schnabel operation than by any other method and when a shortening of the conjunctival surface is evident, it is more surgically correct than a Snellen. With it, it is the rule rather than the exception to obtain complete relief.

In conclusion the position, therefore, is for trachoma in the absence of fresh importations to disappear under better conditions of living, but when it is contracted and treatment delayed, to cause endless trouble to the patient. Its characteristic features are in short its curability as a community disease, but its intractability as an individual affection. In opening this discussion I have attempted to present a reasonable statement of its problems, which I now leave for your consideration.

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Dr. J. Campbell Douglas (Newcastle) said that while the fresh evidence in favour of cell inclusion bodies was stimulating and suggestive, it was not very convincing. Further work in this direction was required and might be carried out by Australian ophthalmic surgeons. The differences in the clinical appearances between trachoma on the one hand and *ophthalmia neonatorum* and gonorrhœal infections of the conjunctiva on the other were against ætiological relation of the two diseases. Its relation to non-gonorrhœal *ophthalmia neonatorum* was discounted by the work of Browning, at Moorfields Hospital, who had found that about 90% of these cases were due to the pneumococcus.

He desired to say something on the standard of cure in trachoma. Many of the patients who had attended for long periods at Moorfields Hospital, had shown little evidence of disease beyond photophobia. McCallam, with all his experience, had expressed to the writer his difficulty in deciding in certain cases whether or not a cure had been complete. The speaker suggested as a standard of cure the following conditions: (i.) There should be freedom from discharge, (ii.) the conjunctiva should be pale and smooth, free from redness and injection and dilated vessels, (iii.) hypertrophied papillæ and follicles should have completely disappeared or should have been replaced by cicatricial tissue, (iv.) pannus should have completely retrogressed or been replaced by scar tissue, (v.) photophobia, lachrymation, pain and discomfort should have disappeared.

Dr. J. Lockhart Gibson said that in cauterising the corneal ulcer of hypopion keratitis he invariably penetrated into the anterior chamber with the finest cautery point until the aqueous oozed out. He did this because he had found that otherwise healing was apt to occur superficially and in such a manner that infective matter was left in the deep parts of the wound; in such circumstances there was a risk of return of hypopion and irritation perhaps after the lapse of a week or more. For this reason he did not believe in conjunctival grafts except for fresh wounds made by the surgeon and known to be aseptic.

He considered acute glaucoma to be due to increased secretion of the ciliary gland just as increased intracranial secretion was due to hyper-activity of the chorioid plexus. He complimented Dr. Paton on his paper.

After it had been well for six months trachoma did not recur. He advised the continuance of 1:5000 corrosive sublimate for twelve months. He could not think that trachoma was very contagious. He had observed many cases in the Brisbane Hospital for sick children during a period of twenty-five years. The children had not been isolated and no child in the hospital had ever contracted trachoma. He was not satisfied that one of the Australian soldiers contracted trachoma in Egypt; the only men he had seen with trachoma in Lemnos and in Egypt had contracted it before they left Australia.

He was not in favour of tarsectomy. Contraction after trachoma was often due to the use of drastic remedies but he would not put carbon-dioxide snow in this category. Syphilis often complicated trachoma. Trachoma was curable if patience was exercised in its treatment.

Dr. F. Antill Pockley expressed his appreciation of the President's paper which contained such a wealth of reference and information that it was impossible to discuss it fully. He had always maintained that there was little or no evidence of the infectivity or contagiousness of trachoma and was pleased to note that Sir James Barrett had come to the same opinion after his experience in Egypt. He agreed that at all points in New South Wales trachoma was a disappearing disease. He attributed this to the fact that the subjects of trachoma now received better and earlier treatment than formerly, lived under better hygienic conditions, and had greater facilities for getting away from the hot climate whence the comparatively few instances of trachoma that they now saw came.

Dr. Pockley expressed the opinion that the incidence of trachoma was largely racial and climatic. Some years ago he had written a thesis upon the subject of trachoma and had circularised the whole profession in Australia. The replies showed that in scarcely any instances had trachoma originated in people who had always lived upon the seaboard; very few examples occurred in the highlands, and practically all came from hot, dry, inland districts. Sir James Barrett had some time before prepared a chart of the incidence of trachoma in Victoria and it showed remarkable agreement with the speaker's chart for the whole of Australia.

For topical treatment there did not appear to be anything better than the time-honoured copper stick and nitrate of silver. Nearly every drug in the pharmacopœia suitable for external application had been tried for trachoma. He had abandoned the use of carbon-dioxide snow because of the after pain and for the same reason he had given up irrigation which he had employed twenty or more years ago. Irrigation had a remarkable effect upon the trachoma, but it was too painful. Such heroic treatment as jequirity and the inoculation of gonorrhoeal pus was never heard of nowadays. He considered tarsectomy a most valuable method of treatment, greatly shortening the course of the disease but he agreed with Dr. Temple Smith that it should be reserved for the chronic stage of the disease.

Some speakers had laid great stress on the use of the common towel as being a means of communicating trachoma from one person to another. Believing as he did that true trachoma was negligibly if at all contagious, he asked how was it that trachoma frequently ran its course in one eye while the other eye remained unaffected? Presumably the patient did not use a separate towel for each eye.

Sir James Barrett referred to the ophthalmia among Turkish prisoners and the position as regards trachoma in Australia.

Dr. Edward Ryan said that he used operative measures, tarsectomy and canthotomy, in advanced trachoma only.

Dr. Leonard Mitchell stated that he had given up the use of carbon dioxide snow as patients would not tolerate the pain.

Dr. E. Temple Smith advised the use of nitrate of silver and operative measures in late stages of the disease only.

Dr. Morgan ascribed the freedom of South Australia from trachoma to generally better social conditions in that State.

Dr. T. A. Price used solid carbon dioxide dissolved in ether and painted on the lids with a brush. Medical men were sent into Western Queensland to educate the public in trachoma.

Dr. W. Wallis Hoare held that tarsectomy was of great help in selected cases and was in favour of extensive peritomy.

Dr. Guy Pockley had used much carbon dioxide snow and had found that patients tolerated it better on hot days.

Dr. M. A. Schalit discussed treatment in the East.

Dr. H. F. Shorney pleaded for the notification of trachoma, for the education of trachomatous children and for better selection of immigrants.

At the conclusion of the discussion Dr. Paton replied briefly.

THE PREVENTION OF MYOPIA.

By James W. Barrett, K.B.E., C.B., C.M.G., M.D., M.S., F.R.C.S.,
Melbourne.

Though a great deal of attention to the causation of myopia has been given, it cannot be said that a satisfactory solution of the problem has been achieved. There seems to be no doubt that myopia is most common amongst people of sedentary habit who persistently use their eyes for near work. The use of the eyes for near work, however, is far from being the only cause. One-sided progressive myopia, so-called "malignant myopia" is familiar to us all.

In 1888 Mr. Lang and I published an account of the refractive character of the eyes of mammalia and showed that 7% of the eyes examined were myopic. The animals which possessed myopic eyes, were with the exception of two monkeys, all domesticated and with the exception of the monkeys possessed practically no accommodation. We then pointed out that it was impossible to assign the cause of myopia in these cases to the overuse of the eyes for near work. There is evidently a constitutional factor which is difficult to define. Wild animals so far as our experience went were never myopic.

Now every oculist is familiar with the child or adolescent who is becoming increasingly myopic. The report of the parents is that the child is always reading, is not very fond of games, but is absorbed in books. You find the myopia increasing about a diopter every twelve months and you know that this increase may continue till say twenty-five years of age. It is true that cases are met with in progressive myopia in which the subjects are not studious, but it can be definitely stated that most of the cases belong to the class indicated.

The question I am submitting for discussion to-day is what can be done to stop this increase. According to the report of the Committee on the Causes and Prevention of Blindness (1922) myopia is responsible for 13.98% of all cases of blindness at all ages. I do not think that in Australia myopia is responsible for anything like so large a percentage, but it is sufficiently serious. It seems to be generally agreed that a myopia of over five diopters must be regarded as serious. If the eye be regarded as approximately a sphere, then any increase in the radius will be followed by enlargement of the surfaces proportionate to the square of the radius and of the capacity of the eye in proportion to the cube of the radius. If then a patient possessed twelve diopters of myopia, the elongation of the eye would be approximately four millimetres. The increase of surface would be as the square of twelve to the square of fourteen and the increase of cubic capacity as the cube of twelve to the cube of fourteen. But as the increase is not distributed over the whole of the eye, but only over the posterior part, the actual damage done is even more serious. Five diopters of myopia may well be regarded as a danger point.

The construction of suitable desks, the proper lighting of schools, and the provision of good print are all factors of value, but experience has shown that these alone effect results of limited value. I think it was Maddox, of Bournemouth, who first pointed out that when you are confronted with children who show signs of progressive myopia, very radical action must be taken if the process is to be stopped. It used to be the practice to instil atropine and send the child into the country, in other words to give up education. The advice was impossible to follow in some cases and apt to be disastrous in others, so far as the future of the child was concerned. It was Maddox, I think, who showed that the essence of the problem was to alter the outlook and habits of the child. The future occupation of such a child should not be a sedentary one and should be preferably one which calls for the constant moderate use of muscle and bone. The trouble arises, however, as to how the necessary education may be effected and Maddox suggested the formation of myopes' schools and or myopes' classes. Twenty-one of these myopes' classes have been created in London. The schools are used for children who possess myopia from five to fifteen diopters. Those with less than five diopters are allowed to follow the ordinary school curriculum with precautions and those over fifteen diopters are taught at schools for the blind. The curriculum is divided into three parts:

(1). Oral teaching with the normal children for such subjects as can be taught orally.

(2). Literary work, such as is necessary for the knowledge of the ordinary means of communication to be learned without books, pens or paper, but by the use of blackboards and chalk, the writing to be done freehand fashion.

(3). A free use of every sort of handicraft that will develope attention, method and skill with the minimum use of the eyes.

The details of the curriculum organized on these principles are set out in *School Hygiene* of March, 1919. It will be seen, however, that it is the principle enunciated by Maddox that is fundamental. The child's inclination must be turned from books to the living world around it and it must be induced to abandon the idea of a sedentary occupation and to take to a business in life which involves muscular activity, at all events in part, in the open air. Fortunately, so far as my experience goes, we have not in Australia so far these appalling cases of child myopia, but we have quite enough myopia and we have no myope classes or myope schools though I urged their institution seventeen years ago. It is for this Congress to say whether the problem should be brought under the notice of the various educational authorities in Australia.

I append an essay by Maddox on this subject, dated 1884.

Short-sight is with certain exceptions and if regarded broadly the penalty of the studiousness either of those who have it, or of their ancestors. It is a natural consequence of the prolonged occupation of the eyes with near objects exacted by the educational and literary requirements of the age. We may say, therefore, that the principal cause of short-sight is short-seeing. It is aided by sedentary habits, vitiated atmosphere and poor nutrition, though the latter tends rather to cause a distinct form of short-sight which occurs among the lower classes quite apart from education and which is happily uncommon.

Short-sight is almost unknown in wholly uneducated nations. Its relative prevalence among the different classes and conditions of men has been abundantly proved to be in direct proportion to the sedentary and confined or studious nature of their employment. Statistics to this effect have been collected in nearly every civilised country.

In this country so long as nearly a century ago a paper was read before the Royal Society to point out the great prevalence of short-sight in the Oxford Colleges as compared with its almost entire absence among the uneducated classes.

At a later date about fifty years ago that very acute observer, Mackenzie of Glasgow, then Queen's Oculist for Scotland, wrote of short-sight as follows: "Rarely congenital, but often connected with the hereditary tendency, myopia owns, as a chief predisposing or exciting cause, the using of the eyes too much in early youth on small and near objects."

It often takes several generations of short-seeing to induce short-sight, and in this way heredity becomes established. Motais has shown that even animals, if imprisoned for several generations, become short-sighted. The tables here reproduced illustrate the influence of heredity. In the New York schools German and American children work together under exactly the same conditions, but short-sight is much more common among the former than the latter for the simple reason that the German children are descended from the most studious of all civilized nations.

In New York Schools.

German children	24 % myopic.
American children	20 % myopic.
Irish children	15 % myopic.
Coloured children	2½ % myopic.

Time would fail me on this occasion to enter into the details of general school hygiene, but I would like to raise one interesting question, viz., whether it might be possible to stay the onward march of short-sight in those families in which it is deepening generation after generation.

I think it would, but only by very radical measures. One of these is colonization, which generally brings a reversion to more primitive conditions of life and often succeeds in resuscitating families that have become overcivilized.

Yet even without leaving this country I have long believed that an immense deal could be done by appropriate educational methods in a school (or in a department of a school) specially set apart for the purpose.

For excellent as have been the measures taken to prevent short-sight in schools scarcely any special provision appears to have been made for children who are already short-sighted. Even children who inherit from their parents the progressive form of short-sight, are still allowed to compete side by side and on equal terms with sound eyes in ordinary schools, where teachers cannot in fairness be expected to alter the modes of education for the sake of a small short-sighted minority.

Since "over-long" sight (hypermetropia) is the natural condition of a young child, the existence of the opposite kind of sight evidences a greater departure from nature than when found in an adult.

But the chief disadvantage of myopia for a child is that the eyes have to run the gauntlet of the dangerous years of education, with the absolute certainty of the short-sight being increased thereby for the rest of life. Were the educational period divested of its dangers, there would be comparatively little to fear in after-life, for happily it is only in the highest degrees that any tendency to progression generally continues after the coming of age. Whatever degree of the defect is reached during youth is retained for the greater part of life thereafter. In such a special school as I propose everything should be adapted to prevent increase of the defect. Books should be used with great moderation and with every precaution as regards suitable paper and type, posture and illumination. Evening work for the eyes should be entirely abolished and the labour be laid upon the shoulders of teachers instead, who should impart knowledge verbally, read aloud and ask questions. It is usually the evening preparation which does most damage to a short-sighted child. If the illumination is imperfect the child bends more closely over the book. Yet the effort to increase the light causes in the case of gas a warm and vitiated atmosphere, which impairs the vital elasticity of the coats of the eye and renders them more prone to the distension in which short-sight consists. It also causes a tendency to weak irritability of the eye and to accommodative spasm which, by shortening the focus of the eye for the time being, makes vision still nearer. The stooping posture thus engendered and which children more readily assume at the close of the day when the muscles of the back are tired, hinders the return of blood and increases the liquid pressure within the eye at the very time when the coats are least prepared to resist it.

To the idea that examination work would be impossible with so restricted a use of books the following experience may give an answer. Two students of equal year entered simultaneously many years ago for what was then the most difficult surgical examination in this country. For the prior six months one was read to entirely by the other without perusing a page himself and passed easily; while his kind friend, through a demonstrator of anatomy, was unsuccessful. It might be difficult to find teachers sufficiently enthusiastic, self-denying and capable, but the demand would no doubt create the supply and after all examinations are more often hindrances than aids to a practical education.

In a special school far more tact and talent would be needed than in ordinary teaching and industry would be as necessary in the teachers as in the taught, since it is the former who really do the work. The expense would be a limitation, though it is just in the upper classes that the defect is most prevalent, and for whom the need is greatest.

It may be objected to the scheme that on leaving school books will have to be read. This is true, but the critical time will be over and wisdom will presumably have been acquired to use them rightly. With it all, we must remember that, when short-sight is already acquired in childhood, some progress in the affection is inevitable, being due to the growth of the eye in common with the rest of the body, but this character of progress is comparatively innocent, and is not accompanied by pathological changes at the back of the eye. Our aim should be to make it a little as possible.

Dr. D. D. Paton in considering defective vision associated with nebulæ differentiated between a pre-existing myopia, an apparently acquired and a pseudo myopia, the latter being the class of case mentioned in speaking of trachoma yesterday.

Meyerhof considers that the high prevalence of myopia in Egypt was due to the corneal opacities caused by trachoma but his experience more resembled that of Steiger of Zurich who after examination of high school children concluded that corneal nebulæ did not cause myopia at all. In troublesome cases there was as great a diversity of refraction as among non-trachomatous cases, but whether the

percentages were different in the two cases it was more difficult to determine, and also one generally did not know the refraction of the eyes before the onset of trachoma. Apart from the nebulæ there might be alteration of curvature from the same conditions that caused the nebulæ.

Re convergence, Sidler Huguenin considered it of no importance, because in anisometropia where binocular vision is impossible the myopia might still increase. Ochi after inserting wedges under the muscles to obtain a pressure on the globe concluded muscle pressure had probably little influence if any in altering the shape of the globe unless the sclera had been already weakened.

Re the amount of correction Sidler Huguenin, he thought, had published figures which showed 73% of slightly undercorrected cases were stationery, 63% of strongly undercorrected cases were stationery, so that with both methods of relief there was no great alteration in prognosis.

Bishop Harman had found so low a degree of vision in 84 out of 99 cases of myopia above 10 diopters that he had placed it second as a cause of practical industrial blindness after senile cataract. Rush placed it as the 4th cause in China.

He was satisfied that there existed a class of myopia which was as it had been succinctly phrased, phylogenetic and not ontogenetic, and was due to a mesoblastic defect or instability. Nothing would prevent the development of this type except improving the general condition of the patient. He has found above 5 diopters of myopia in each eye in about 17% of 400 myopic patients.

At the Hospital for Sick Children in Perth the State Government maintained a teacher for the trachomatous children detained there.

Dr. T. R. Muirhead (Adelaide) said that from a consideration of notes of many cases and from personal observation it did not appear that in South Australia at all events myopia was sufficiently prevalent to justify an appeal or recommendation to the Government for the establishment of special schools.

ACQUIRED AXIAL MYOPIA.

By F. A. Newman, M.D., B.S. (Melbourne), D.O. (Oxon),
Melbourne.

Myopia is that dioptric condition of the eye, in which with the accommodation at rest incident parallel rays of light come to a focus at a point anterior to the light sensitive layer of the retina.

Only that form due to increased length of the eyeball, which generally developes in early school life, namely acquired axial myopia will be considered; this increased length affects the posterior pole of the eye; the part anterior to the equator may be normal. It is a disease of civilization. It is rare in new-born infants. Among uncivilized people and among civilized people one of the chief factors in its causation is prolonged work requiring close vision.

In this article an attempt will be made to discuss some of the effects of civilization in its causation.

It will be necessary to consider a few anatomical and physiological points.

Accommodation.

The act of accommodation is due to the alteration of the shape of the lens, brought about by the contraction of the ciliary muscle. This muscle is attached anteriorly to the inner surface of the sclerotic inside the posterior end of Schlemm's canal and also partly to the lamellæ of the *ligamentum pectinatum* at the angle of the anterior chamber. This anterior attachment of the muscle may be regarded as its more or less fixed point.

It has three sets of fibres, circular, radial and meridional.

During accommodation the following events occur.

The lens becomes more spherical than when the lens is at rest. When accommodation takes place the radius of curvature of the posterior surface stays about the same, whilst that of the anterior surface becomes six millimetres, so the lens becomes practically a sphere.

The lens in the early decades of life is plastic and conforms to the shape of its elastic capsule. It is anchored to the ciliary body by the suspensory ligament In the condition of rest, accepting Helmholtz's theory of accommodation, the fibres of this ligament and the lens capsule are tense, but when the ciliary muscle acts during accommodation, these fibres and the capsule of the lens are relaxed and the lens becomes spherical.

The posterior surface of the lens is supported by the jelly-like vitreous, so the relaxation of its capsule is greater on its anterior surface; the lens becomes bowed forwards into the less resistant aqueous of the anterior chamber. This causes the anterior chamber in the lenticular region to become slightly more shallow.

There is a question raised here. Is the practical non-alteration in curvature of the posterior surface of the lens due entirely to the viscidity of the vitreous? It will be suggested later that it is not the only factor, the other being compression of the vitreous due to pressure caused by tension of the elastic layer of the chorioid.

The relaxation of the suspensory ligament and lens capsule, is caused by contraction of the circular fibres of the ciliary muscle. This draws the ciliary processes towards the equator of the lens, but at the same time the lens becomes more spherical.

The pupil contracts. This causes a flattening out of the iris; the mouths of the iris crypts are opened; there is less tucking of the iris towards the angle of the anterior chamber, consequently a more easy exit of the aqueous, by the iris crypts and Schlemm's canal.

The ciliary muscle is partly attached to the sclerotic, to the inner side of the posterior end of Schlemm's canal and to the posterior fibres of the *ligamentum pectinatum.* When it contracts, the action of its circular and radial fibres would tend to draw inwards, the inner wall of Schlemm's canal thus widening it, and by this means allowing the more easy exit of the aqueous during accommodation.

By the contraction of the meridional fibres the chorioid is made tense, hence it is known as the *tensor chorioidae.* It is said that the chorioid is only affected in its anterior portion. This seems improbable, as when the eye is dissected, the chorioid is seen to be attached firmly to the sclera only where it ends at the optic nerve entrance and less firmly at the exits of the *venae verticosae* and entrances of the ciliary arteries and nerves. Over the rest of its external surface there is only loose connective tissue.

It is suggested that the whole of the chorioid is made tense, but of course the effect is lessened by the *venae*

verticosae and to a less extent. at the points of entrance of the ciliary arteries and nerves.

The effects of tension of the chorioid would be compression of the vitreous.

If the whole of the elastic layer of the chorioid is made tense, and *venae verticosae* are drawn slightly forward, thus allowing the more easy exit of blood and, if the vitreous is compressed, the supra-chorioidal space is enlarged, thus allowing more room for the dilated blood vessels of the chorioid when the eye is accommodating.

The Elastic Structure of the Eye.

The outer coats of the eye are lined on their inner surface by a definite elastic membrane formed by the secretion of cells. Descemet's membrane lines the posterior surface of the cornea. It is derived from the endothelial cells covering its inner surface. At the angle of the anterior chamber this membrane splits into fibres which pass into the trabeculæ of the *ligamentum pectinatum.* They are covered with endothelial cells continuous with those lining Descemet's membrane.

The fully developed *ligamentum pectinatum* consists of two parts: (a) An outer more compact part lying in apposition with the sclerotic and forming part of the inner wall of Schlemm's canal and (b) an inner looser part, which persists in man until the sixth month of fœtal life, when it disappears and with it much of the spaces of Fontana.

Posteriorly the *ligamentum pectinatum* enters the ciliary body and iris and is continuous with their elastic tissues.

The ciliary body contains many elastic fibres. They are gathered together in the outer part of its base into circular and oblique bundles and are continuous with the *ligamentum pectinatum.* On the inner side they join the continuation forward of the elastic membrane of the chorioid, the *lamina vitrea* or membrane of Bruck. In the ciliary body this membrane splits into two layers, an outer which joins the *ligamentum pectinatum,* and an inner under the retinal pigment epithelium. This is continued along the posterior surface of the iris forming its posterior limiting membrane or *dilator pupillae.*

The inner surface of the chorioid is lined by an elastic membrane, the *lamina vitrea.* When stained with rosin this membrane is a perfectly homogeneous structure, but when stained with Weigert's elastic tissue stain, is seen to be composed of two layers, an inner homogeneous and an outer finely reticular. At the margin of the papilla they end differently, the inner ceasing with the pigment epithelial layer and the outer continuing on for some little distance into the substance of the papilla.

These two layers have a different origin, the inner one being formed by a secretion from the cells lining it, the retinal pigment epithelium. This layer is formed during embryonic life.

The outer reticular layer is continuous with the elastic fibre net work of the chorioid.

The lens is surrounded by an elastic capsule formed by a secretion of its epithelial cells.

The suspensory ligament of the lens is said to be derived from the hyaloid membrane, which separates the vitreous from the retina, passing in front behind the lens. Where it lies on the ciliary processes to which it is closely adherent, it sends off fibres which pass radially from the apices of the ciliary processes to the lens capsule, forming its suspensory ligament. There are three groups of these fibres: (a) anterior inserted into the anterior part of the capsule, (b) middle inserted into the equator, (c) posterior inserted into the posterior part of the capsule.

It would seem there are certain objections to the above view of the origin of the suspensory ligament from the hyaloid.

(i.) The ciliary processes are lined by the retinal pigment epithelium, so that the fibres are attached to epithelial cells. It is questionable if this attachment would be strong enough to stand the strain put upon the fibres.

(ii.) The lens with its suspensory ligament may be compared to a suspension bridge in which there are three sets of ropes, upper, middle and lower, corresponding to the anterior middle and posterior fibres of the suspensory ligament. The attachment of the ropes of the bridge to the posts are widely separated for mechanical reasons. Now the apices of the ciliary processes are opposite the equator of the lens, so apart from the objection of attachment to epithelium, the question of mechanical advantage arises.

It is suggested that the suspensory ligament is derived from a continuation inwards of the *lamina vitrea* and *ligamentum pectinatum* where they join in the ciliary body.

If there is anything in this view the mechanism would be better, more like the ropes of a suspension bridge and again there would be continuity of the elastic structures inside the eye.

This view is rather favoured by comparison with a bird's eye. Normally a bird's eye like that of man is focussed for distant objects. Accommodation for near objects is accomplished in both by increasing the convexity of the anterior surface of the lens.

In man, this is brought about by relaxation of the suspensory ligament, in birds, by drawing back of the posterior lamina of the cornea, where it breaks up into the *ligamentum pectinatum.* The retraction of this ligament is due to a special muscle (Crampton's muscle) which corresponds to the ciliary muscle in man, but differs from it in being a voluntary and not an involuntary muscle.

The sclerotic contains many fine elastic fibres mingled with the connective tissue fibres running in the same direction as these fibres and more numerous in the outer than inner layers.

That the elastic lining of the eye is capable of standing considerable pressure is evidenced by the development of a Descemetocele in some cases of ulceration of the cornea.

The primary changes in the formation of an aneurysm are associated with interference of the nutrition of its elastic lining. It is suggested that the primary changes in myopia are associated with interference of the nutrition of the elastic lining of the eye, that the bulging of the sclerotic is secondary and as long as this elastic lamina is healthy, myopia will not develope.

Excessive Accommodation.

It has been suggested that during accommodation the whole of the elastic lamina of the chorioid is tense and the vitreous compressed. The more work put on the ciliary muscle during accommodation by bringing work too close

to the eyes, the greater the strain on the elastic lamina of the chorioid. An emmetropic eye in a child of ten has an amplitude of accommodation of fourteen diopters, that is its near point in seven centimetres.

In a series of observations made on children between eight and twelve years with normal distant vision it was found many of them brought close work too near to the eyes, the average distance between the eyes and the work in those who did was eight centimetres; that is they were using twelve diopters of accommodation, only having about two diopters of reserve. It is estimated that only 60% of the accommodation should be used, so as not to call up the reserve of the ciliary muscle.

When a muscle is worked to its full capacity, it quickly tires and when working thus, it may strain the tissues it is pulling on. So when the ciliary muscle is working to its full capacity for long, one of two things may happen.

(1) The muscle tires; the child gets headache or other symptoms of eye strain and is brought for examination. Refraction shows slight hypermetropia. By rest from close work for a time, if necessary, by resting the ciliary muscle by atropine and attention to the general health and by a change and suitable diet the muscle recovers its tone and by regulation of the amount of close work and the conditions under which it is carried out, the child goes on well and no harm is done to the eyes.

An objection is here raised to the acceptance of this theory of excessive accommodation being a cause of myopia, owing to the fact that hypermetropes have to exert excessive accommodation, even when doing close work at the normal distance. Undoubtedly many hypermetropic children become myopic. Whether they do or not probably depends on the presence of congenital or acquired weakness of the elastic membranes within the eye. If this weakness is absent, the child gets symptoms of ciliary muscle strain; this is corrected by wearing suitable glasses and doing close work under suitable conditions.

(2) The elastic lamina within the eye is unable to stand the tension and is strained, probably it stretches a little first.

The eye posterior to the lens is somewhat dome shaped, the apex of the dome being about the macular region. This would be the part affected first. It is suggested that the primary change in myopia is a slight stretching of the elastic lamina in this region, thus increasing the antero-posterior diameter of the eye. By this means the work of the ciliary muscle is lessened, but the child still goes on working too close and too long and the membrane stretches more and more, increasing further the antero-posterior diameter of the eye and myopia is established. As in aneurysm, once the elastic tissue is stretched, its nutrition is interfered with; it is then unable to stand the normal intra-ocular pressure and tends to be stretched more and more.

If it progresses, ultimately the stretched membrane atrophies; splits may occur in it; the chorioidal vessels atrophy; the atrophic changes at the macula, and temporal edge of the disc follow. This may explain the crescent at the temporal edge of the disc. When any tissue atrophies, it is replaced by a certain amount of scar tissue. Probably when the elastic lamina splits and the chorioid atrophies, the split edges of the elastic lamina are bound down to the sclerotic firmly and as the sclerotic stretches, the lamina will not give in this region where it is bound down by scar tissue, so as the posterior pole of the eye enlarges, the chorioid and retina on the nasal edge of the disc are drawn over this edge forming the supertraction crescent. Once the inner elastic lamina stretches and splits, the outer sclerotic coat, losing the support of the elastic lamina, quickly stretches and the myopia progresses.

As the posterior pole of the eye enlarges and the antero-posterior diameter of the eye increases, less and less work is thrown on the ciliary muscle. It atrophies until ultimately accommodation is not used at all, the near work being held at such a distance from the eye as to form a conjugate focus with the macula. So the relation between accommodation and convergence is upset.

The fundus changes bear no definite relationship to the degree of myopia, but, generally speaking, the higher the degree of myopia, the greater are the fundus changes.

Why should the elastic lamina give in some cases of excessive accommodation and not in others? There probably is some primary malnutrition of the elastic structures within the eye. The splits which occur in Descemet's membrane and posterior capsule of the lens in some advanced cases cannot be explained by a mechanical theory.

Children of myopic parents probably have congenitally weak elastic tissues of the eye. But there are many in whom there is no family history of myopia. In these acquired weaknesses there probably is some malnutrition of these elastic structures and the strain of close work under bad conditions starts the cycle.

Possibly myopia may be a nutritional disease allied to rickets and be associated with the absence of vitamins in the food. Unfortunately many infants are now reared solely on the much advertised dessicated artificial foods from which these vitamins are absent.

Broadly stated it may be said that any interference with parental or post-natal nutrition of a child predisposes to the development of myopia. Consequently it is important to attend more carefully than is the rule to the nutrition of the prospective mother, especially the myopic mother.

If the infant cannot be naturally fed, the food should be as near the natural article as possible.

If there is anything in the theory of excessive accommodation being the exciting cause, then care of the eyes during school life is very important. The tendency in modern schools is to let a child do close work too early. It would be a wise rule if all children below twelve were taught more by distant vision and the sense of sound, near work only in moderation and no home lessons.

The formation of desks is important. They should be inclined towards the child at an angle of about 10° to 15°, the edge of the desk being at a level about midway between the umbilicus and lower end of the sternum. When writing and reading the left elbow and forearm should rest on the desk between the book and edge of the desk in such a position that the arm is almost vertical. Then it is impossible for the child to sprawl over his work. If, in spite of this, the child gets too close, the vision should be at once tested and if found normal, a head rest fixed to the desk should be used when doing close work.

Writing or reading in the above position gives an average distance in a child of seven years twenty-three centimetres, in a child of nine twenty-six centimetres and in a child of eleven twenty-eight centimetres. Periodical inspection of children's eyes is important and those whose vision is not normal or who have symptoms which may be associated with eye strain, should be refracted under atropine.

Unfortunately it is rare to get a patient in the early stages of myopia. Usually the child is brought, because the writing on the black-board cannot be seen from the desks. By this time myopia has begun. In a few cases it has been my lot to watch myopia develope. A child is brought whose static refraction under full atropine cycloplegia is one diopter, when the effect of the atropine is gone this child is practically emmetropic. After a time the child is examined again and myopia has developed.

What should be done with these children? If we accept the view that the primary lesion is in the elastic layer of the chorioid and that the exciting cause of the lesion is due to excessive tension of that membrane, the treatment indicated would be rest of that membrane and the ciliary muscle. The only way this can be accomplished is by avoidance of close work. To make sure that accommodation is not used, it would be wise to keep the eyes under atropine for a few weeks, so as to rest the ciliary muscle and the elastic layer of the chorioid. This gives them time to recover their tone. The effect of the atropine should be allowed to go off gradually, by lessening the strength of the drops, so as to make sure that the ciliary muscle will gradually return to work, the whole process occupying about two months.

Those children with static refraction of say one diopter may be regarded as on the road to myopia; its developement may possibly be prevented.

If the child has already a low grade of myopia, the same treatment is suggested with the addition of glasses correcting the myopia.

To sum up, the treatment suggested in such cases is:

(1) Absolute avoidance of close work for about six months, one month under full atropine, a second month under modified atropine.

(2) Absence from school during that period, a change to the country and attention to general nutrition by diet, fresh air and exercise.

(3) After return to school, careful regulation of the amount of close work and the conditions under which it is carried out.

There can be little doubt that the success of a school is now-a-days judged too much by examination results, the children have too much close work and too often in doing it the accommodation is taxed beyond its reserve.

Even supposing all this is wrong and that excessive accommodation is not a factor in the causation of myopia, surely it is not wise to allow children to overtax their ciliary muscles by bringing their work too close to the eyes. The average school hours is five. Children above the age of nine are given home lessons as well. It is quite common to see a child of about fourteen or fifteen with symptoms of eye strain who has home lessons which take about two or three hours. This means that the eye is accommodating about seven to eight hours each day and this hard work occurs just at the period when the child is entering a new epoch in life, a period during which it is essential to attend to the physical rather than the mental developement of the child. How can we expect the generative organs to develope satisfactorily when at this critical age the child is in many cases studying hard for examinations? In girls it is especially essential that excessive study should be abolished during such an important period, thus allowing the generative organs to develope satisfactorily and so in after years lessening the terrors of child-birth and also putting the young mother in a better position to nurse her infant naturally.

CLINICAL DANGERS INTRODUCED BY THE WASSERMANN REACTION AND THE USE OF SALVARSAN.

By J. Lockhart Gibson, M.D. (Edinburgh), M.R.C.S. (England),
Brisbane.

It has occurred to me that this subject is one which should be discussed in our Section. Ophthalmologists see more reason for diagnosing syphilis, at least tertiary syphilis, than those practising in any other department of medicine. They are able, also, to observe more definitely the results of treatment and to clinch by the effects of treatment the clinical diagnosis arrived at.

We will probably all agree that a failure to react to the Wassermann test before treatment cannot be relied upon as evidence that the lesion is not syphilitic and that a failure to react to the Wassermann test following treatment does not justify the conclusion that the patient has been cured of syphilis.

Our knowledge of syphilis has increased greatly since the introduction of the Wassermann test, which is, when it yields a positive response and even occasionally when it fails to do so, a very valuable aid to diagnosis, though probably a greater danger than safeguard to the patient after treatment has been established. The same applies, I think, to arseno-benzol preparations and to intensive treatment with them. They have taught us much and are a valuable aid in the treatment of syphilis, though I would say less of an aid in ophthalmic practice than in any other department of medicine.

They have, however, introduced an attitude of false security in the case of both doctor and patient, which results in harm instead of good to the patient. I know that medical men who pin their faith to intensive treatment with arseno-benzol, insist that they are not satisfied with that drug and a course perhaps of intensive mercury also, that they follow on with mercury for years. But either owing to want of insistence or persistence on their part or to want of perseverance on the patient's part the intensive portion of the treatment is apt to be the only part thoroughly conducted; and when followed by one or more failures to respond to the Wassermann test, it results in the patient and often the doctor concluding that he is cured and can go on his way rejoicing. Does not the ridiculous demand of our health authorities cherish this delusion? They ask for a certificate of "cure."!!

The poor patient is, indeed, perhaps not even in as good a position as the man who comes to us after the age of fifty or sixty years, who gives no history of syphilis, and has had no treatment, but who, we know, has acquired it in his youth and has fought the primary and the secondary infection himself by his own specially good powers of tissue resistance, as well as or better than any short course of intensive treatment could have done. He fails to show any symptoms until he begins to go down the hill and may have been the father of healthy children or of many healthy and one diseased one.

Such patients, as we know, often react to mercurial treatment in a most satisfactory manner and some of them live to advanced old age without seeking or even requiring treatment.

It has been urged that there ought to be ophthalmic physicians as well as ophthalmic surgeons. In my opinion we should call ourselves ophthalmologists. If we are not both physicians and surgeons we have no right to practise ophthalmology.

I would like to quote from the August, 1923, number of the *Proceedings of the Royal Society of Medicine*, what Dr. Farquhar Buzzard said on this subject to the Section of Neurology. He says:

"We have never had and are still without reliable data on which to base the opinion that any particular individual has been cured of syphilis. . . . In spite of treatment . . . late manifestations are still frequent. I submit that we shall never obtain better results, until we frankly admit that we cannot justifiably use the word "cure" in relation to syphilis and unreservedly support the principle of lifelong treatment. . . . Is it fair to ourselves or to our patient to judge by the persistence of a negative Wassermann reaction and the propagation of healthy children? Clinical experience is full of evidence to the contrary. I submit that despite its undoubted scientific interest, the Wassermann reaction is an obstacle rather than a help in the practice of medicine. A positive reaction is sometimes a valuable piece of confirmatory evidence. A negative reaction is almost without value as evidence, but unfortunately it has been so highly credited that it is a direct menace to proper treatment."

Such opinions from a neurologist of experience are worthy of attention. My own observations and study largely confirm them. I must, however, admit that when an initial clinical conclusion is supported by a failure to react to the Wassermann test it encourages one's belief in the absence of syphilis and a reaction to the Wassermann test often helps one.

It is impossible to avoid the suspicion regarding the action of "Salvarsan" that, while it is apt to get rid of manifestations of syphilis very quickly, it may lead only to the retreat of the spirochaetes into entrenched or encysted positions, from which they are even more ready to become active in the future, than if the treatment had been a thorough course of mercury. No doubt the fact that mercury when used alone (unless by injection) is continued for a much longer period, may be chiefly answerable for this. It seems to me that neurologists and ophthalmologists are more likely to agree in the matter of the treatment of syphilis than those engaged in other departments of medicine, because the cases seen by both of them are more often of a tertiary nature.

The following three cases will illustrate my contentions.

I. U. J., aged 30 years, contracted syphilis in July, 1917. He had intensive treatment, *id est*, injections of "Salvarsan" and of mercury during seven weeks shortly before he left Europe; his serum then failed to react in the Wassermann test. He was told to have another Wassermann test in February, 1918. When in the boat coming back to Australia in October, 1917, *id est*, three months after infection, his left eye went blind after paining. When seen by me in January, 1918, the pupil was occluded and there were complete dense permanent looking posterior synechiæ. The eye counted fingers at sixty centimetres (two feet). Mercurial inunction and atropine resulted in commencing improvement in three weeks and in the counting of fingers at 3.6 metres (four yards). I do not know what became of him afterwards. He had had no anti-syphilitic treatment after leaving England until I saw him. His statement that he was to have another Wassermann test in February, 1918, had protected him from treatment on the boat or more correctly condemned him to no treatment.

I cannot recall a case of a single returned man among the eye cases seen by me in Brisbane, who had been treated intensively for seven weeks and whose serum did not yield a Wassermann reaction, who was instructed to follow up with mercury, even by the mouth. They were as a rule, however, told to have a Wassermann test carried out after some months' interval.

II. A. J. H., aged 31 years, was seen on June 16, 1922. He gave a history of pain in his right eye for six days of lachrymation and defective sight. He had never had trouble before. The vision of his right eye was $6/_{24}$ and of his left eye $6/_5$. His eye was deeply injected, his iris discoloured; there were complete posterior synechiæ and some deposit in the pupil. No history of syphilis could be elicited, but a history of gonorrhœa in 1916 which lasted for not more than three weeks and was not followed by a gleet. He was married; his wife was healthy; he had one child, three years old and well. His wife had had no miscarriages. He had not had rheumatic fever. He had rheumatic pains lately. Clinically the eye was suffering from syphilitic iritis. I sent him to have a Wassermann test done that day and also sent him to bed to begin inuncting that day with 3.6 grammes (sixty grains) of *unguentum hydrargyrum* and, of course, to use atropine. The following day Dr. Duhig reported that the serum did not yield a Wassermann reaction, but that the complement fixation test for gonorrhœa was positive. I telephoned to continue inunction, but ordered also 1.2 grammes (twenty grains) doses of sodium salicylate and 2.4 grammes (forty grains) of sodium citrate every two hours night and day. This was on Saturday. Both treatments were continued until the Monday when I visited him. The eye was dramatically improved. No pain, practically no injection; the pupil was fully and equally dilated; his vision was $6/_{18}$ The improvement in three days was almost too quick for inunction, although not more than I expect in four days. So taking that and the failure to react to the Wassermann test together, I foolishly stopped inunction and continued with atropine locally and with salicylate and citrate of soda. He was kept in bed. On subsequent visits further improvement was not noted in spite of the removal of some diseased teeth. I stopped the salicylate on July 14, 1922, a month after seeing him first. I had wasted a month except for the first three days. Vision had then retreated to less than $6/_{60}$, and although the pupil kept fully dilated, the iris was again discoloured and a little injection persisted. Furthermore, I found numerous vitreous opacities, not particularly fine ones. He was immediately sent back to bed and inuncted. Seven days later vision had improved to $6/_9$ partly. There was no injection and the vitreous was almost clear. After a further four weeks the eye saw $6/_6$ and a month later still $6/_5$. Inunction was continued; not of course with confinement to bed.

III. X. O. W., aged 32 years, contracted syphilis in May or June, 1921. In January and February, 1922, he had five injections of arseno-benzol, followed by injections of grey oil until November, 1922. He was then given mercury and iodide by the mouth and latterly only iodide of potash. There was no response to the Wassermann test in April, 1922, August, 1922, November, 1922, and January, 1923. The right eye began to trouble before Christmas of 1922, that is, before the last Wassermann test. When seen first by me on April 19, 1923, I found the vision of the right eye fingers at ten centimetres (four inches) only, and of the left eye 6/5 partly. There were signs of iritis but very numerous fine membranes and sand-like vitreous opacities, also one or two coarse opacities. He was put to bed and kept there and made to inunct with 3.6 grammes (sixty grains) *unguentum hydrargyrum*. In two weeks the eye counted fingers at 3.6 metres (four yards). At the end of a third week vision was 6/60, at the end of the seventh week 6/9 (one letter). The vitreous was practically clear except for two coarse opacities which suggested that they would persist. The disc was seen to be recovering from neuritis and the veins were distended. He is continuing daily inunction with a break every seventh day and latterly without confinement to bed. After the first month 1.2 grammes (twenty grains) doses of iodide of potash three times daily were given, also for some weeks. The vision is now 6/9 partly. He is fat and well and has gained over three kilograms (half a stone) in weight. The optic neuritis has subsided. The remaining two coarse opacities are pretty central, and will probably prevent a vision of more than 6/9 being obtained.

I have an idea that there may be at least three attitudes to this little effort of mine. Some will think it reactionary. Others will think that it contains merely their own accepted beliefs, others that improvement under mercurial treatment does not prove an affection to be syphilis. They are views held by me for years and freely expressed to my colleagues in Brisbane. I have thought that they would provoke useful discussion.

DR. J. C. DOUGLAS said that failure of the patient's blood serum to react to the Wassermann test on a single occasion was no proof of the absence of syphilis. Repeated tests, including perhaps one after a provocative dose of 0.45 gramme of "Salvarsan," were necessary to decide the question. Discrepancies were due to the personal equation among laboratory workers, to differences in the antigens employed and to daily variations in the strength of the reacting substance in the serum of the patient.

A failure on the part of the serum to react might mean the encapsulation of spirochaetes in syphilitic foci, which might at a later period break down and induce a return of the power of serum to react. In this connexion the sigma test was in some respects a better test than the Wassermann. A consistent failure to react to the Wassermann test of the serum after treatment and over a period of two years might be accepted as evidence of cure. He could subscribe only partially to Farquhar Buzzard's views in this matter. Syphilitic infection was a factor in so many diseases that the whole question was one of great importance.

SIR JAMES BARRETT said that a reaction of the patient's serum to the Wassermann test was of the utmost importance, while a failure to react was comparatively valueless. It appeared to him that if their criterion of cure were the response to this test, some patients were incurable, since treatment by arsenic and mercury failed to alter the power to react, which had been described under these circumstances as "Wassermann fast." He felt that some instances of supposed sympathetic ophthalmia were syphilitic and were not really connected with the injured eye. The reduction in the number of cases of sympathetic ophthalmia reported in recent years was probably due to better knowledge and also to the advantage taken of the Wassermann test. He had seen patients in whom a diagnosis of sympathetic ophthalmia had been made and later had to be revised as syphilis.

DR. J. LOCKHART GIBSON in reply said that when he was sure the eye lesion was syphilitic, he did not have the Wassermann test carried out, because in the event that the serum did not react the patient might thereby be influenced not to persevere with treatment. The results of treatment depended greatly on the fighting powers of the patient's tissues against the spirochaetes and their effects. In military and repatriation work a Wassermann test was invariably required if syphilis was suspected, but as a rule then it was stated at the outset that treatment must be maintained even if the report should be that there was no reaction. In the intractable types mentioned by Sir James Barrett it was just possible that thyreoid administration might help. He was careful in choosing his pathologist for the performance of the Wassermann test and always asked for the ice-box method.

In answer to Dr. Paton Dr. Lockhart Gibson said that the serum of children affected with interstitial keratitis always gave a reaction to the Wassermann test. He had found that the serum of those children to which he had the Wassermann test applied, lost its power to react after treatment.

SYMPATHETIC OPHTHALMIA.

By J. LOCKHART GIBSON, M.D. (Edinburgh), M.R.C.S. (England),

Brisbane.

THE subject of sympathetic ophthalmia still requires elucidation. Two cases I am about to report and the suggestions from ophthalmic literature from other sources indicate that the explanation of many if not all of these may be (i.) serious injury to one eye, (ii.) a predisposing cause, for instance, a state of the general health, such as a constitutional taint, acquired or inherited, or deficient activity of one or more of the ductless glands, especially of the thyreoid or possibly the existence of some focal infection. Together these determine an attack of plastic inflammation in the sound eye. Is it not possible that injury to one eye without some such predisposing state will not result in sympathetic inflammation?

I am bringing to your notice the history and results of treatment of two cases to illustrate constitutional syphilis as a cause of what would, as a rule, be diagnosed as sympathetic inflammation of the sound eye.

One of these patients I have watched since 1899; the other I have watched for a year. It gave me perhaps the most serious fright in my experience, until I was able to put it down to inherited syphilis.

After giving the essential features of these two cases, I propose, if you permit me to do so, to indicate that interstitial keratitis is in some particulars parallel to sympathetic ophthalmia.

W. M., aged forty-two years, had his right eye injured by a piece of steel on January 31, 1899. He said that the

eye was removed in Queensland about seven weeks later, but that it was not taken out soon enough and affected the other which became nearly blind. He said that Dr. Maher had treated it for three weeks before removal and then advised removal, but on returning to his doctor in Queensland he was advised at first to keep it. It then became worse and was removed within a week. When I saw him first on August 11, 1899, he had been returned from Sydney some six weeks, where he had been under Drs. Evans and Odillo Maher for three months. The eye had improved under treatment. The Sydney doctors said that they had never seen a case of the sort that did so well. The good left eye had been fairly well until after the injured eye was removed.

He was advised to put himself under my care. I found vision 6/9; there were many posterior synechiæ and some punctate deposits on the posterior surface of the cornea. No vitreous opacities were present as far as could be seen through a small pupil. He had been treated in Sydney with atropine and mercurial inunction and had continued both until four weeks before I saw him. He stated that his pupil got quite round and big while in Sydney. I doubt this. There was evidence, however, when I saw him of fresh iritis. The posterior synechiæ were so firm and established that I failed to break them down except very partially with persistent use of atropine.

He had been allowed to discontinue mercury, no doubt on the supposition that the eye had been suffering from sympathetic inflammation and that it had subsided. My diagnosis then and one I think confirmed by his subsequent history and past history was that the left eye had suffered from syphilitic iritis induced in a syphilitic subject by the injury to the other eye.

The treatment by mercurial inunction had resulted so favourably, not because it had cured a case of sympathetic ophthalmitis, but because it had benefited a case of syphilitic iritis. The iritis was recrudescing when I saw him. He first denied any history of venereal trouble, but on pressure admitted that he had had a chancre in 1880 which lasted for a month. I found palpable cubital glands. His wife was strong; she had borne him five strong children and had no miscarriages.

I instructed him to continue inunction and atropine and told him my diagnosis. I saw him again a year later; he had discontinued inunction, and there was some returning iritis. Inunction was recommenced. Vision corrected was 6/6. A year later he consulted me again, namely, in 1901. Vision with correction was 6/5. The adhesions were holding and the pupil consequently was small. He continued inunction until 1902. When seen in 1904 vision was still 6/5, but the eye had been aching. He was instructed to resume inunction and did so. I did not see him again until 1913 when his eye had been paining him for six weeks and vision less good; "a cobweb in front of his eye." His vision was 6/9 (partly); there was evidence of slight iritis.

He was instructed to resume inunction for at least a year. When seen a year later vision was 6/5 and he had gained over three kilograms (seven pounds) in weight. He continued inunction for a further seven months. When seen last in 1917 at the age of sixty his vision was 6/6 (partly) and he looked well. There was nothing fresh in the eye, but he was advised to resume inunction for a few months.

I have reported this case at length and for the first time. You may not all agree with me that the eye was a syphilitic one. I am satisfied myself that it was and that reported cases of sympathetic inflammation in which recovery has occurred after mercurial treatment, were of this nature.

P. C., aged 8 years, was referred to me by Dr. Robina Harlin, of Killarney, on September 14, 1922. Five weeks previously she was pulling an old-fashioned bow and arrow and the arrow came back and hit her right eye.

I found the eye injected; the scar of a penetrating wound was opposite the upper edge of a medium-sized pupil. The iris was discoloured. A broad anterior synechia of the upper edge of the iris to the scar was seen. The lens was becoming opaque. A one millimetre hypopion was present in the anterior chamber. The tension of the eye was increased. The eye had perception of light. I advised that the safest course would be enucleation. I consented to try and save the eye. An incision was made with a Graefe knife and an iridectomy was performed. The hypopion with turbid aqueous fluid was evacuated. The hypopion did not re-form, but the coloboma gradually filled with lymph. Tension kept low after the operation for three weeks and then came up to natural. The eye appeared to trace light, but I was not happy about it because of the lymph in the coloboma. I, therefore, kept her under observation. The other eye continued to see well and the injured eye was cosmetically good and quite comfortable. On December 5, that is nearly three months after I first saw her and after an absence of more than a week I found the left or good eye a little injected. The pupil was irregular and sluggish; the vision was 6/6 (partly). Posterior synechiæ were very numerous and demonstrated by atropine. The adhesions broke down and the pupil became large and round with continued use of atropine. Needless to say that these findings had the effect on me of "putting the wind up." If "sympathetic" is meant blindness for the child and enormous regret for me. I put the child at once into a private hospital and began mercurial inunction that day. I also enucleated the right eye that day. I found that the child following this one had been born dead at seven months and was said to have been dead for a month. Also that this child had convulsions as a baby, but only once. My only hope was that the iritis in the good eye was syphilitic. The fact that the coloboma had filled with lymph in the other eye could also be explained on a syphilitic basis. The injection of the eye cleared up as soon as the posterior synechia broke down. Dr. Duhig examined the blood and reported that it gave a very strong Wassermann reaction.

Inunction has been continued and the eye sees well and only shows marks on the anterior capsule of the lens where the iris margin was attached.

It does not appear to me to be stretching a point to bring interstitial keratitis into this connexion. As honorary ophthalmologist to a large children's hospital for fully twenty-five years, seeing my own out-patients as well as my own in-patients, I have had a very large number of patients with interstitial keratitis under my care. I wish to raise my voice, not for the first time, against the still very prevalent teaching regarding these cases. My own experience in Brisbane has taught me that they are a manifestation and to the patient a very useful manifestation of inherited syphilis. They are this whether the blood serum yields a reaction to the Wassermann test or not. I have found, indeed, that the result of the test is almost uniformly a reaction. Interstitial keratitis occurs not only in young subjects of inherited syphilis, but also in adults of thirty years. In adults a history of a determining cause, such as trauma, or of some long standing strain, such as active service, can generally be obtained. In the case of children a history of trauma as an immediately determining cause can also occasionally be obtained. In such cases very energetic treatment by mercurial inunction will safeguard the second eye from a similar attack, if commenced early enough. It does so also in some cases in which no history of trauma can be

obtained. This leads me to suggest as an explanation of those cases in which the second eye becomes attacked during treatment, that the treatment was either not begun soon enough or was not energetic enough. Inunction has not only to be used, but it has to be used thoroughly. "Salvarsan" has appeared to me to help inunction in some cases and in others not at all.

I believe that anti-syphilitic treatment is the only treatment for interstitial keratitis. Atropine applied locally is necessary in some cases to control the *sphincter iridis*. I have found that uveitis is a very rare complication of interstitial keratitis, unless, of course, anti-syphilitic treatment is not carried out. Did I not hold these opinions, I would still employ energetic and prolonged anti-syphilitic treatment in a case of interstitial keratitis because by doing so I would be treating a dangerous constitutional state fraught with grave danger to the future life and health and usefulness of the patient. The teaching that anti-syphilitic treatment is not the treatment of interstitial keratitis is to me so pernicious that it is difficult to speak of it calmly.

Sir James Barrett advised that in dealing with apparent sympathetic ophthalmia very careful inquiry should be made into the venereal history of the patient.

Dr. A. M. Morgan stated that all his patients in the Adelaide Children's Hospital affected with interstitial keratitis yielded a positive finding when the Wassermann test was applied to the blood serum.

Dr. F. Antill Pockley said that he had had very little experience in sympathetic ophthalmia. He related the history of a patient who had met with an accident by which he got a piece of brass or copper in the eye. The medico-legal aspect became an issue and expert witnesses had contended that the removal of the eye was wrong, but he held strongly that the eye should have been removed.

Dr. Lockhart Gibson reminded the members that at the last Congress the Section of Ophthalmology had affirmed the desirability of removing such eyes.

Moved by Sir James Barrett and seconded by Dr. Lockhart Gibson: That the Federal Committee be informed that the Section of Ophthalmology resolved unanimously as to the following definition of blindness for the purposes of compensation and that the definition be forwarded to the various Governments with a view to amending existing acts, and that Insurance Companies be informed:—

"That total loss of sight in an injured eye be defined as follows (for the purposes of assessing compensation). Total loss of sight in an injured eye is defined as vision in that eye with or without glasses, of less than counting fingers at two metres, and the vision of such a character as to furnish no help in the occupation of the patient."

ARTERIO-SCLEROSIS: OPHTHALMIC SIGNS AND SYMPTOMS.

By J. Campbell Douglas, L.R.C.P. & S. (Edinburgh), L.F.P.S. (Glasgow), D.O. (Oxon),
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Definition of Arterio-Sclerosis.

Sir Clifford Allbutt says that arterio-sclerosis is not a disease but a lesion common to many diseases; if we accept this view we will have a clearer insight into its nature and pathology.

That a man is merely as old as his arteries is an old and well known proverb. My own definition of arterio-sclerosis is that it is a chronic inflammatory thickening of the arterial coats with or without degeneration, diffuse or circumscribed.

Varieties of Arterio-Sclerosis.

Here again I like best the clinical classification of Clifford Allbutt:

(1). Hyperpietic arterio-sclerosis, associated with hyperpiesia and either due to it or both due to the action of a common pressor stimulant, perhaps an excess of adrenalin in the system from deranged functioning of endocrine glands or to general bodily strain or again, to combined physical and mental strain as in so many business men or possibly as one of the consequences of errors of refraction.

(2). Toxic arterio-sclerosis, due to such toxæmic processes as gout, syphilis, alcohol, tobacco, lead, constipation, pyorrhœa and similar toxæmias and without any increased blood pressure.

(3). Decresent or senile arterio-sclerosis, due to senile or involutionary changes, generally with a moderate increase in blood pressure.

Others, however, adopt a classification based on histological characters, such as nodular, diffuse, hyperplastic, senile, fatty and hyaline. I believe that it is better to think in clinical terms and I am sure every case of arterio-sclerosis can be fitted into one or other of the above classes. There are, of course, cases which overlap from one type into another.

Pathological Nature of Arterio-Sclerosis.

Arterio-sclerosis is a form of chronic arteritis and the essential lesion is a proliferative thickening of the *intima* from activity of the endothelial cells and also of the sub-endothelial layer which shows great increase of connective tissue. The medial coat may show either muscular hypertrophy as a physiological response to increased blood pressure or an inflammatory fibrous overgrowth with later degenerative changes. There is a considerable increase of the elastic tissue which may come from growth and splitting of the internal elastic lamina, or may possibly arise from endothelial cells. The adventitia may be thickened, but this is of minor importance. The various changes in the arterial wall are cell proliferation, increased elastic tissue, formation of connective tissue and production of hyaline and fatty substance and all these may result from increased activity due to irritation and, except the last perhaps, are not degenerative.

Degenerative changes play but a secondary part in arterio-sclerosis. They are present chiefly as fatty changes in the *intima* and *media*, caseation in the *media* and calcification on the inner surface of the vessel and sometimes in the perivascular sheath. All degenerative changes in arterio-sclerosis are of late onset and are subsequent to the inflammatory changes and will not be recognized in early cases. While syphilis equally with other general dyscrasia such as tuberculosis and gout may cause a typical arterio-sclerosis, yet the histological changes in the vessels in the former are generally different and distinguishable

from those in the latter. It is interesting to compare them. In syphilis the pathological process commences in the *adventitia* and is mainly expended there. The *intima* is usually the seat of extensive proliferative changes, but the connective tissue and elastic fibres have not the orderly arrangement of arterio-sclerosis. The vessels have a pearly appearance, round or cylindrical, with no calcification or casestion on their surface. While syphilis and tuberculosis have so many points in common, it is noteworthy that the former plays an infinitely more important rôle in disease of the blood vessels. Finally I would say that senile arterio-sclerosis is more often degenerative than proliferative and that here closure of capillaries by hyaline deposits or fatty changes is a prominent feature. The vessels walls, too, are more ready to give way leading to subcutaneous, to cerebral or to retinal hæmorrhages.

Diagnosis.

With this I am chiefly concerned. It may be divided into early symptoms and early signs. Early symptoms are asthenopia, failing vision, transient amblyopia, presbyopia of early onset, *amaurosis fugax*, giddiness on rising or lying down quickly, headache without obvious cause such as sinus trouble or astigmatism, insomnia, digestive disturbances, intermittent polyuria. The majority of these symptoms suggest eye trouble and lead the patient to consult the oculist who is thus enabled to make an early diagnosis long before the physician can do so. The physician is accustomed to look for late symptoms before making a diagnosis, such symptoms as dyspnœa, palpitation, *angina pectoris*, cramp and pain in the calves and ankles in walking, drowsiness in the morning, loss of memory, inability to concentrate, transient attacks of hemiplegia or aphasia, migraine, neuralgia and nervousness, the Stokes-Adams syndrome.

We are not concerned with these here. When they are present the disease is advanced and little can be done for the patient. It is the early diagnosis on I wish to insist. Having discovered the symptoms, one looks for the signs. Early signs of arterio-sclerosis are found mainly in the eye and mainly in the *fundus oculi*. They are abnormal recession of the near-point of vision, sluggish pupil reaction, narrowing of the arteries with a fullness of the veins, the proportion between them being as two to four instead of the normal as three to four. You never see a nice fat artery in arterio-sclerosis. Pallor of the fundus is present, as are deepening of the colour of disc, blurring of the disc edges, loss of translucency in the vessels, a broadening and brightening of the light reflex on the arterioles, a slight beading of this reflex, a disappearance of the light reflex on the veins for a short distance on either side of its crossing by an artery, a sudden darkening of the vein on either side of a crossing as if it were sunk in a little pit; the veins cross beneath the arteries more nearly at right angles like a beam of light which has undergone refraction, resuming their original course when clear; sometimes the vein takes a slight U-shaped bend or lateral displacement. These are early signs. None of them are very definite as you will notice; no single one or two or even three suffice to make a diagnosis; they are easily overlooked unless the vessels are systematically examined in every direct ophthalmoscopic examination. They will not be seen in every vessel but usually only in some. Taken together, however, they form the clinical picture of arterio-sclerosis at that early stage at which it can be treated with success. Late signs would be silver-wire arteries, pipe-stem sheathing of arteries, small white exudates, generally unilateral, small hæmorrhages, here and there along the course of the vessels, thrombosis of a branch with corresponding scotoma in the field of vision. compression of veins with banking of the distal blood stream; in a word the clinical picture of an arterio-sclerotic retinitis, together with other bodily signs and perhaps a cerebral hæmorrhage. When the patient gets to that stage, there is no need to call in the oculist; the physician can make the diagnosis himself. In a recent work called "The Oxford Medicine" this statement occurs:

> The arterial disease is always far advanced before it is detected. Medical science has not yet advanced sufficiently far to be able to recognize this condition in its early stage nor the circumstances that predispose or cause its onset, except in such a disease as syphilis. Nay more, we do not yet understand the steps which should be taken to acquire this knowledge.

To my mind the steps are clear. Call the oculist in consultation for he and he alone has the means of recognizing arterio-sclerosis in its earliest stages from a direct study and inspection of the blood vessels under a high magnification; those very vessels, the arterioles, which are the first and principal vessels to be affected. Of course, if you agree with my friend, Dr. C. O. Hawthorne, of London, that no man is worthy to be called a physician who cannot use his ophthalmoscope with as much ability and confidence as any oculist, then there is no more to be said. But how few physicians have Dr. Hawthorne's skill and opportunities with that technical instrument?

Conclusion.

To sum up, arterio-sclerosis is a condition which can be diagnosed more surely and at an earlier stage by the ophthalmic surgeon than by anyone else. The signs and symptoms which the physician is accustomed to depend on are late signs and symptoms. If the physician who suspects this condition in a patient, will refer him to the oculist for report, the latter will not only confirm the diagnosis at a stage at which its actual root cause can be sought and eradicated, but in addition will know the actual stage to which the condition has progressed.

Sir James Barrett congratulated Dr. Douglas on his paper. He felt, however, that the earliest reliable ophthalmoscopic sign of arterio-sclerosis was the nipping of the veins. The widening of the light streak and the relative size of artery and vein he found unreliable as evidence of arterio-sclerosis. In order of time of appearance and of importance the signs were (i.) nipping of the veins, (ii.) hæmorrhages, (iii.) "silver-wire" arteries, (iv.) white effusion. The last mentioned was in his experience of the gravest importance. He pointed out, however, that very high blood pressure (240 millimetres of mercury) might not be accompanied by any changes in the *fundus oculi*. The nipping of the vein was due to the thickening of the artery. He agreed that the earliest signs of arterio-sclerosis were available to the oculist, but if they were absent, he must not conclude that there was no increase of tension.

Dr. D. Paton said that subjective phenomena had been

described as earlier evidence of arterio-sclerosis than objective findings, but these photopsias *et cetera* were in his opinion of value only if correlated with the ophthalmoscopic abnormalities. He thought the slight bending of the vessels of paramount importance and this was often corroborated at an early stage by increased colouration in the disc. He had found red free light of assistance in the detection of linear hæmorrhages.

The early findings of bending of the vessels was of great value in guiding the physician in the treatment of the middle-aged patient with obscure headache. It was necessary that oculists should cooperate with physicians in the attempt to establish as an entity if it be possible the fundus of hyperpiesis. This condition was regarded by Sir Clifford Allbutt as curable in some instances as distinguished from arterio-sclerosis.

A CLINICAL STUDY OF ONE HUNDRED AND TEN CASES OF RENAL DISEASE AND ARTERIOSCLEROSIS.

By Leonard J. C. Mitchell, M.D., B.S. (Melbourne), *Honorary Ophthalmologist, Melbourne Hospital.*

It has been well said that it is not possible to draw a hard and fast line between renal retinitis and arterio-sclerosis and any set of observations which will help towards the forming of a diagnosis in doubtful cases, cannot fail to be of interest to ophthalmologist and physician alike.

The underlying pathological condition of the renal type is to be found in the kidney and the retinal changes in these cases must vary with the type and intensity of the nephritis. It must not be assumed, however, that the changes in the retina give the key to the type of kidney lesion and, further, it must be borne in mind that arterio-sclerotic retinitis may be accompanied with or succeeded by signs of renal retinitis.

Also it is necessary to note that P. H. Adams has described a typical renal retinitis caused by a severe toxæmia due to a septic wound.

Haemorrhages.

The present view is that the hæmorrhages come from the capillaries by diapedesis and not by rupture, the toxic spoiling of the vessel walls being caused by the poison circulating in the blood. The microscopically demonstrable changes in the media and intima of the retinal vessels may be due to the same cause.

Exudates.

The typical cotton wool patches have been proved to be fibrinous and they infiltrate extensively the layers of the retina in a fine fibrinous meshwork. The star figure shows a large amount of hyaline exudate in Henle's layer; the rods and cones appear to have completely degenerated. Later large globular phagocytic cells appear and absorb the hyaline masses. Though individual cotton wool patches disappear fresh areas are developing.

With regard to retinal lesions in different types of nephritis it is not possible to draw sharp lines of distinction, but broadly it may be stated that (i.) acute nephritis excluding pregnancy shows retinitis late in the disease. Moore, however, in five out of one hundred and nineteen cases of trench nephritis noted ophthalmic changes coming on in from five to thirteen weeks after onset. (ii.) Chronic parenchymatous nephritis is associated in 25% of cases with eye changes. (iii.) Chronic interstitial nephritis without uræmia shows retinitis in 50% of cases with uræmia in 75%.

As regards prognosis this is affected definitely as soon as retinal changes take place. Nettleship's contention that few if any live longer than two years, still holds good for general purposes.

A review of the table shows several points of clinical interest. The patients were all in-patients at the Melbourne Hospital.

Acute Nephritis.

Eight patients were examined; none died; two had uræmia; one had a retinal hæmorrhage with a blood pressure of 100 millimetres; one had papillœdema, but the condition was complicated by old osteo-myelitis. The blood urea was above normal in one patient only.

Sub-acute Nephritis.

Ten patients were examined; three have died. The heart was enlarged in five patients; one had uræmia, five patients had either retinal œdema or hæmorrhage. Three had increased blood urea.

Chronic Nephritis.

Fifty-one patients were examined; fifteen have died. Sixteen had cardiac failure and sixteen had uræmia; twenty-three had blood pressure over 160 millimetres and urea concentration was below normal in twenty-two. Fundus changes were numerous; fifteen had arterio-sclerosis; twenty-five had papillœdema; eighteen had hæmorrhages and eighteen exudates.

Chronic Nephritis with Arterio-sclerosis.

Twenty-one patients examined; ten have died. Sixteen had blood pressure of over 160 and eight over 200 millimetres. These eight all showed severe retinal signs (œdema, exudates and hæmorrhages) and four died. Sclerosis of retinal vessels was observed eleven times, hæmorrhages and œdema fourteen times and exudates eight times.

The total fundus findings number eighty-three patients in one hundred and ten or 75.4% which is very much higher than most observers admit. Even excluding the fifteen patients with arterio-sclerosis in whom no hæmorrhages, exudate or papillœdema were found, the percentage is 61.8. Only five of the twenty-one arterio-scleroties had normal urine.

The twenty-eight patients with exudates had an average blood pressure of 190 millimetres; the forty-two with hæmorrhages had an average blood pressure of 185 millimetres; and the forty-four with papillœdema had an average blood pressure of 178 millimetres of mercury.

The result of the urine examination is shown in the table, as are the cardiac conditions. Albumin was present in the urine of all but sixteen patients.

I have to acknowledge the valuable work of Drs. Ethel Parnell, M. C. Gardner, F. J. B. Miller and G. A. Owen in assisting in making the fundus examinations and Dr. G. A. Penington's enthusiastic work in collecting the medical findings from the records of the Melbourne Hospital.

Sir James Barrett congratulated Dr. Mitchell on his paper. He had found that retinal changes were practically unknown in acute nephritis, rare in sub-acute and

ANALYSIS OF 110 CASES OF RENAL DISEASE AND ARTERIO-SCLEROSIS

No.	Age	Sex	Diagnosis	Blood Pressure Millimetres	Heart	Urine				Blood	Fundus Oculi				Result of Wass. Test	Remarks
					Lesions.	Alb'min	Casts	Pus	Urea Contn.	Urea	Sclerosis of Vessels	Exu-date	H'ges.	Papil-loe-dema.		
1	15	F	Cn.N.	160/120	+	+	+	-	-	-	-	-	-	-	-	-
2	54	F	Cn.N.	190	+	+	+	-	-	-	-	+	+	+	-	-
3	51	F	Cn.N.	126	N	+	+	-	-	-	-	+	+	+	0	-
4	47	F	Cn.N.	260	A2	+	+	+	-	-	-	+	+	+	-	-
5	66	F	Cn.N. C.F.	260/100	+	+	+	-	-	-	-	+	-	+	-	-
6	48	F	Cn.N. U.	200/130	+	+	+	-	-	-	+	+	-	+	0	Pda 2 months later.
7	25	F	Cn.N. U.	170/100	N	+	+	-	-	-	-	+	-	+	0	-
8	39	M	Cn.N. C.F.	110	N	+	+	-	-	-	-	+	-	+	0	-
9	65	M	Cn.N.	—	-	+	+	-	-	-	+	+	-	+	0	-
10	54	M	Cn.N. A.S.	190	+	+	+	-	-	-	+	+	-	+	+	-
11	59	M	Cn.N.	170/70	+	+	+	-	-	-	+	+	-	+	+	-
12	65	F	Cn.N. C.F.	190	+	-	-	+	-	-	+	-	-	-	+	-
13	62	F	Cn.N. U.	150	-	-	+	+	-	-	+	-	-	-	+	-
14	58	F	Cn.N. U.	130	-	+	-	+	-	-	-	-	-	-	+	-
15	65	M	Cn.N. C.F.	170	+	+	-	+	-	-	-	-	-	-	+	Died.
16	56	F	Cn.N.	124/80	-	+	-	+	-	-	-	-	-	+	-	-
17	49	M	Ch. A.S.	245/100	+	+	-	+	-	-	-	-	-	+	-	-
18	52	F	Cn.N.	170/135	+	+	-	+	-	-	-	+	+	+	-	-
19	49	M	Cn.N. A.S.	188/110	+	+	-	-	-	-	+	-	-	-	-	-
20	58	M	Cn.N. U.	238/136	+	+	+	-	-	-	+	-	+	-	-	-
21	?	F	Cn.N.	138/68	-	+	+	+	-	-	-	+	-	-	-	-
22	77	F	Cn.N. C.F.	—	-	-	+	-	-	-	-	-	-	-	-	Cataract. Died.
23	62	F	Cn.N. C.F.	150	-	-	+	+	-	-	-	-	-	+	-	-
24	47	M	Cn.N. U.	110	-	+	+	-	-	-	+	+	+	-	-	-
25	59	M	Cn.N. A.S. U.	222/137	N	+	+	-	-	-	-	-	-	-	-	-
26	39	M	Cn.N. A.S. C.F.	170	-	+	+	-	-	-	+	+	+	+	-	Died.
27	68	M	Cn. N. C.F.	fibrillating	+	+	+	-	-	-	+	+	+	+	-	Died
28	53	F	Cn.N. A.S. C.Hg.	215	+	+	+	-	-	-	-	-	+	-	-	Died.
29	47	M	Cn.N. C.F.	152/68	+	+	+	-	-	-	-	+	-	+	-	-
30	41	F	Cn.N. C.F.	160/100	-	+	-	-	2.85%	-	+	-	+	-	-	-
31	20	M	Cn.N. U.	170/140	A2	+	+	-	-	19mg	-	-	-	+	-	Died.
32	57	M	Cn.N. U.	235/100	-	+	+	-	-	-	-	+	+	-	-	-
33	65	M	Cn.N. C.F.	160	-	+	+	-	-	-	+	-	+	+	-	Dissemd chorioiditis.
34	19	F	Cn.N. U.	80	+	+	+	-	-	-	-	-	-	-	0	Died, small pale kidney.
35	33	M	Cn.N. A.S. C.F.	238/180	+	+	+	-	-	-	-	-	+	-	-	Retinal oedema.
36	74	M	Cn.N. A.S. C.H.	200	+	+	+	-	-	-	++	-	-	-	-	-
37	52	M	Cn.N. C.F.	90	+	-	-	-	-	-	++	-	-	-	0	-
38	30	M	Cn.N. A.S.	240/170	A2	+	+	-	1.45%	88mg	-	-	+	+	0	Died, total nitrogen 8.8gm.
39	37	M	Cn.N. A.S.	130/92	+	+	-	-	-	-	-	+	+	+	0	Died, pneumonia.
40	58	M	Cn.N. A.S. U.	180	+	+	-	-	-	-	-	+	+	+	-	Died.
41	50	M	Cn.N. A.S. U.	240/150	+	+	+	+	1.85%	-	++	+	-	-	-	Died.
42	47	F	Cn.N. U.	140	-	+	+	+	-	-	+	-	-	-	-	-
43	26	F	Cn.N. U.	140/100	+	+	+	-	2.2%	51mg	-	-	-	-	0	-
44	47	M	Cn.N.	120/90	-	+	+	+	3.15%	33–62mg	-	-	+	+	-	Persistent low blood pressure.
45	78	F	Cn.N. A.S. U.	110/85	+	+	+	+	-	-	++	+	-	+	-	Died.
46	53	M	Cn.N. U.	170/110	+	+	+	+	-	-	-	+	+	+	0	Died.
47	45	M	Cn.N. A.S.	210/140	+	+	-	+	1.2%	-	-	+	+	+	0	-
48	46	M	Cn.N. A.S. U.	210/140	+	+	-	+	1.375%	-	-	+	+	+	0	Died.
49	14	M	S.N.	130	+	+	+	-	1.55%	20–43mg	-	-	-	-	0	-
50	22	M	A.N. U.	182/110	+	+	+	+	-	-	-	-	-	-	0	Streptococcus.
51	18	M	A.N.	90	-	+	+	+	-	-	-	-	-	-	-	-
52	28	M	S.N.	146	+	+	+	+	-	-	-	+	+	+	-	Died.
53	18	F	S.N.	90	+	+	+	+	-	-	-	-	+	-	-	-
54	33	M	S.N.	—	+	+	+	+	-	-	-	-	-	-	-	-
55	15	F	S.N.	—	-	+	-	+	-	-	-	-	+	-	-	Died.
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ABBREVIATIONS :—Cn.N.—Chronic nephritis. C.F.—Cardiac failure. U.—Uraemia. A.S.—Arterio-sclerosis.
A.N.—Acute nephritis. S.N.—Sub-acute nephritis. C.H.—Cerebral haemorrhage. O.—Negative Wassermann reaction.

Analysis of 110 Cases of Renal Disease and Arterio-sclerosis

No.	Age	Sex	Diagnosis	Blood Pressure Millimetres	Heart Lesions.	Urine				Blood Urea	Fundus Oculi				Result of Wass. Test	Remarks
						Alb'min	Casts	Pus	Urea Contn.		Sclerosis of Vessels	Exu-date	H'ges.	Papil-loe-dema.		
56	18	M	A.N.	135	+	+++	+	+	2.2%	50–61mg	–	–	–	–	O	Early chorioido-retinitis.
57	43	M	S.N.	134	–	+++	+	+	1.45%	52–66mg	–	–	–	–	–	—
58	41	M	S.N.	—	–	+++	–	–	–	–	+	–	–	+	–	Died.
59	17	F	A.N.	150/90	–	+++	+++	+++	2.5%	–	–	–	–	–	–	—
60	14	M	A.N.	140	–	++	+++	+++	1.7%	–	–	–	–	–	–	General oedema.
61	33	M	A.N.	100	+	+	+	+	1.4%	–	–	–	+	–	–	Septic broncho-pneumonia.
62	62	M	A.S. C.F.	180/120	+	+	–	–	–	–	+	–	–	–	–	Died.
63	56	M	A.S. C.F.	160/110	+	+	–	–	–	–	+	–	–	–	–	—
64	69	M	A.S. C.F.	—	–	+	–	+	–	–	–	–	–	–	–	Old cyclitis.
65	38	F	Cn.N. A.S.	180/100	–	–	++	–	–	–	–	–	+	++	+	—
66	20	F	A.S.	188/110	–	–	–	+	–	–	–	–	–	–	–	Cystitis.
67	55	M	A.S. U.	190	A2	–	–	–	2.15%	30–32mg	+	–	–	–	O	Senile maculae.
68	62	F	A.S.	180/125	A2	–	–	–	–	–	–	–	–	–	–	—
69	61	M	A.S.	190/90	–	–	–	–	–	–	+	–	–	–	O	Chronic myocarditis.
70	56	F	A.S. C.F.	190	+	–	+	–	–	–	+	–	–	+	–	—
71	49	M	A.S. C.F.	160/90	+	+++	++	–	–	–	+	–	–	–	–	Died, aortic regurgitation.
72	58	M	A.S. C.F.	210/120	A2	+	–	–	–	–	–	+	+	+	Partial	—
73	61	F	Cn.N. C.F.	135/60	–	–	–	–	–	–	–	–	–	–	–	Died. Thick radials.
74	56	F	Cn.N. C.F.	?	A2	+++	–	–	–	–	–	+	–	++	–	—
75	71	M	A.S. C.F.	140/100	–	+	–	–	3.5%	34mg	++	–	+	–	–	—
76	48	M	A.S.	170	+	?	–	+	–	–	+	–	–	–	O	Two fits.
77	45	F	A.S.	250/138	+	+	–	–	–	–	++	–	+	–	–	—
78	40	F	Cn.N. A.S.	174	A2	+	–	–	–	–	+	–	–	+	–	Died, cerebral haemorrhage.
79	68	M	Cn.N. C.F.	120/100	+	+	++	–	–	–	–	–	–	–	–	—
80	62	M	Cn.N. U.	190/110	? +	+++	?	–	1.05%	212mg	++	–	++	+	+	Died, albuminuric ret. and ret. circinata.
81	45	M	A.S.	138	–	+	?	–	–	–	++	–	–	–	O	Cerebral haemorrhage.
82	38	F	A.S. C.N.	156/106	+	+	–	–	–	–	++	–	–	–	+	Angina.
83	78	M	A.S. C.N. U.	150	? +	–	–	–	–	–	++	–	+	–	+	Syphilis.
84	21	M	Cn.N. U.	220/170	–	+	–	–	–	53mg	–	+	+	+	+	Died. Syphilis.
85	60	M	Cn.N. U. Gout	98	+	+++	++	–	1.15%	22mg	+	–	–	–	–	Died.
86	51	F	A.S.	230/110	++	+	–	–	–	20mg	+	–	–	–	O	—
87	25	F	A.N. U.	130	–	+	++	–	2.15%	23mg	–	–	–	+	–	Old osteo-myelitis.
88	35	M	S.N. U.	180/110	+	+	–	–	1.2%	104–63mg	–	–	+	+	O	—
89	36	F	Cn.N.	210	+	+++	++	–	1.45%	59mg	–	–	–	+	O	Abortion, 4½ months.
90	15	M	A.N.	180/110	–	++	++	+	1.5%	31mg	–	–	–	–	–	—
91	31	F	Cn.N.	180	+	+++	–	–	0.85%	68mg	–	–	–	+	–	—
92	26	F	S.N.	120/70	–	+++	++	+	2.2%	35mg	–	–	+	–	–	—
93	25	M	Cn.N.	170/120	–	+	?	–	3.5%	46mg	–	–	–	–	–	—
94	61	F	Cn.N.	100	+	+++	+++	–	–	269mg	+	–	–	–	–	Died.
95	23	M	Cn.N.	120/60	–	++	–	++	1.85%	58mg	+	–	–	–	Partial	—
96	56	M	A.S. C.F.	210/140	+	+++	–	–	3.75%	37mg	++	–	–	–	Partial	—
97	75	M	Cn.N. A.S. U.	200	+	non	e obtain	ed	–	–	+	–	+	–	–	Died.
98	49	M	Cn.N. A.S. C.F.	190/100	+	+	+	+	1.52%	50mg	+(L)	–	+(R)	+(R)	O	—
99	67	M	Cn.N. C.H.	136	–	–	–	–	–	111mg	+	–	–	–	O	Died.
100	63	M	Cn.N. U.	180/130	A2	+	–	–	1.36%	133mg	–	+	+	+	O	—
101	63	F	Cn.N. C.F.	90	+	+	++	–	–	–	–	+	+	+	O	Died.
102	52	F	A.S. C.F.	200	+	+++	?	–	3.25%	23mg	++	–	+	–	O	—
103	72	M	A.S. C.N. U.	200	+	+	–	–	2.65%	71mg	+	–	+	+	–	Died.
104	45	M	A.S.	180	–	–	–	–	–	–	–	?	+	+	–	D.T.'s
105	53	M	Cn.N. C.F.	130	+	+++	–	–	3.65%	65mg	–	–	–	–	O	General oedema.
106	48	F	A.S.	110/98	–	+	–	–	1.2%	44mg	++	–	–	–	–	Died, malignant breast.
107	14	M	S.N.	—	–	+	–	–	1.4%	35mg	–	–	–	–	+++	Chorioido-retinitis.
108	73	M	A.S. C.H.	110	–	+	–	–	–	–	–	–	–	–	–	Died.
109	38	F	Cn.N.	200/160	A2	+++	–	–	0.172%	150mg	–	++	+	+	O	—
110	47	M	Cn.N.	220/155	+	+++	+	–	1.65%	91mg	–	++	–	++	–	—

Abbreviations :—Cn.N.—Chronic nephritis. A.N.—Acute nephritis. C.F.—Cardiac failure. S.N.—Sub-acute nephritis. U.—Uraemia. C.H.—Cerebral haemorrhage. A.S.—Arterio-sclerosis. O.—Negative Wassermann reaction.

far more common in chronic nephritis. The question arose whether retinitis in many patients was not associated with high blood pressure. He had seen a few patients with retinitis without higr blood pressure, but the majority were affected with arterio-sclerosis. Was the kidney condition in such cases part of a general cardio-vascular change? He had been astonished to find how rarely diabetics had retinitis. White effusion represented the terminus of a process and was probably wide-spread throughout the body and disastrous.

Dr. J. Lockhart Gibson agreed that the nipping of veins in the fundus by the arteries was the most important sign and that it was due not so much to raised blood pressure as to hardness of the arterial wall. He thought that physicians should as a routine examine the fundus. They would soon learn to recognize departures from the normal. The normal fundus varied enormously within physiological limits, just as the intra-ocular tension of normal eyes varied within wide limits. It was important to refer patients with arterio-sclerosis to a physician.

Dr. Lockhart Gibson spoke of the importance of emptying the uterus at once in the event of albuminuric retinitis of pregnancy, because the prognosis was then good. He had only seen one patient who had lived for more than eighteen months after the discovery of severe albuminuric retinitis with white exudation; that patient had had only slight changes around the macula. He hoped that the custom of referring such patients to a physician for examination of the urea concentration in the blood and urine might lead to prolongation of life.

Dr. D. D. Paton wished to congratulate Dr. Mitchell on his pains-taking investigation which recalled the care and accuracy of his earlier researches. The question had been raised as to the value of the vascular appearances as an indication of the prognosis. He regarded this as of paramount importance. He had found in consultation with physicians that Nettleship's limit of two years as a safe guide when white exudation was present, but when the fundus picture was of the vascular type, he was inclined to give a better prognosis than formerly. They required to distinguish between the several grades and between those cases in which the fundal changes were secondary to the renal and those in which the retinal vessels merely revealed evidence of a wide-spread vascular change and the albuminuria was a mere coincidence.

Dr. J. C. Douglas asked whether some of the patients with retinitis were suffering from pure arterio-sclerotic kidneys. The details bore out the contention of Geoffrey Evans that a blood pressure of one hundred and eighty millimetres of mercury indicated serious disease. He had recently seen a patient with white retinal effusion, star figure at the macula and moderate optic neuritis. The condition looked like albuminuric retinitis, but no albumin had been found in the urine. A case like this demanded further investigation of the renal function. He congratulated Dr. Mitchell for his excellent paper.

POTENTIAL OR ACTUAL ACCIDENTS AT SEA, POSSIBLY DUE TO VISUAL DEFECTS.

By James W. Barrett, K.B.E., C.M.G., M.D., M.S., F.R.C.S.,
Melbourne.

The late Edward Nettleship in 1913 published an account of cases of accident by land and sea which were due to defective vision. Under the head of shipping accidents he quoted cases that were definitely known to be due to defective eyesight, including two that were reported by me, and in addition he reported a number of cases in which (a) visual defects although present, were not proved to have caused the accident, or (b) such defects, if present, would have explained the accident, but their presence was neither proved nor disproved.

I forwarded him particulars of cases some of which fell under category "a." They were cases in which there were visual defects and there were accidents or mistakes, but definite scientific proof of causation was lacking. Between the interval of my posting him the information and the arrival of the information in London he passed away, so that the cases were not placed on record. I now propose to state them.

One very capable officer was sent to me by a shipping company and was found to possess gross colour defect. He subsequently retired from the sea-faring life. Although he was incapable of appreciating a red light as such, he had successfully navigated ships for some years and the surprise of the marine superintendent on being shown the grave nature of the defect was interesting. Subsequently I became acquainted with the Master Mariner under whom he had served, who gave me the following particulars.

He stated the officer was most capable and that until just before I examined him he had no suspicion that there was anything wrong. He was anchored, however, outside a port into which entry could only be effected at high tide. He told the officer to call him when the coloured signals indicated that he could enter. He called him twice and on both occasions the signals were against him. On the ship's return to Melbourne he was sent to me for examination, with the result I have indicated. The special interest of this case is the length of time the officer had conducted his work successfully without disaster. It takes a combination of circumstances to produce a disaster. It is further a striking comment on the injustice of allowing a sailor to embark on a sea-faring career without a thorough examination.

In the second case a steamer ran aground in a river, two officers being on the bridge. One officer had been rejected by an oculist for colour vision. A second oculist advised re-examination, because he could find nothing wrong, but a little weakness in the colour sense. The second officer had normal vision, but was very slow over his colour tests. The report on this officer from an oculist contained the following passage:

> His answers with coloured glasses show similar results (as the wools) and that though he named a red light correctly, he gave answers which led me to feel sure that he would have difficulty at sea in distinguishing faint green lights from white lights. I do not think he is likely to take a red for a green light, but in thick weather I think he might readily mistake a green for a bright light. I must leave it to your experience of the sea to estimate what amount of risk this may represent.

It is, however, stated that the lights they were steering by were not coloured and it is suggested that the officer who was actively navigating the ship, only possessed 5/9 vision and misjudged the distance.

There is no absolute proof that the grounding of the

ship was due to these visual defects, but the evidence is suggestive.

The third case is the following. A ship grounded at an entrance to a harbour. The channel has a bright light on the one side and it is necessary for the navigator to estimate the distance from the light. The officer in charge was being examined yearly instead of every five years because of doubt respecting his vision. He had mixed astigmatism of two diopters in each eye, one diopter of hypermytropia vertically and one diopter of myopia horizontally. His vision varied, sometimes being down to 6/24 in the right eye and letters of 6/12 in the left, sometimes rising to 6/8 in the right and letters of 6/6 in the left when he pressed on the globes with the lids. He could bring the vision of the left eye promptly to 6/6 in the left when he pressed on the globes with the lids. which he rested against the lower lid. Here again the case is suggestive, but lacks definite proof of causation. by pressing on the globe through the lower lid with the top of his finger. He said that he always used binoculars

Conclusion.

It will be seen that the second and third cases are not conclusive. The unsatisfactory part of marine accidents is that the vision of those who are in charge, is not invariably examined after an accident. In the case of the Victorian Pilot Service the proper course is followed. If a pilot is absent from illness or meets with an accident, he must undergo a thorough medical and ophthalmic examination. Our business, however, should be to urge the authorities in general to make sure that these accidents are humanly speaking impossible. If every boy were submitted to a thorough ophthalmic examination before he went to sea, there would be very little trouble. The value of the periodic examinations was well exemplified lately when an officer was found to be suffering from early tobacco blindness from which he rapidly recovered but he was promptly suspended until his colour vision was normal.

VISION AT SEA.

By D. D. Paton, M.A., M.B., Ch.B. (Edinburgh), D.O. (Oxon),
Honorary Ophthalmic Surgeon, Perth Hospital.

In the absence of any personal experience of marine accidents due to defeceive eye-sight and that of most ophthalmologists must be the same, I have examined the minutes of the evidence of the Department Committee on Sight Tests, 1912. Although twelve witnesses were examined on the point, only one had had under his notice accidents, such as collisions, due to defective form or colour vision. These were four in number and as Manager and Secretary of the Hull Steam Trawlers' Mutual Insurance and Protecting Company, Limited, the witness had dealt with an average of one hundred collisions per year for twelve years. Of these four two were elderly men who may have had defective vision, but in the other two the possible cigarette smoking assigned as a cause on circumstantial evidence cannot be admitted as proven.

Sir Walter Howell had never heard of the question of sight being raised either at a Board of Trade casualty inquiry or in the Admiralty Court.

Lindo Ferguson related two instances in which the defects in vision might easily have proved sources of danger and Sir Walter Howell also mentioned one case where the master of the barque *Peru* averted a collision only in time by correcting the mistake of the look-out man who had miscalled a green light red. He also called attention to another case reported by Captain Heasley.

It is strongly suggested in the report that the lack of evidence of defective sight may be due to the absence of any examination of the officer of a watch or the man on look-out duty at the time in question, not that the question is intentionally burked, but because of the unreliability of the evidence as to the attending circumstances at the time of the accident, such as the distance of lights, objects *et cetera*.

What happened to the master of the *Peru*, (1894) may happen many times, but it may not be reported. Yet the question as to what is the safety limit of colour and form vision is difficult to determine as Freeland Fergus suggested, who, quoting a report of August 31, 1894 (Board of Trade) covering a period of fifteen months shows the rejections affected six men holding second mate certificates who had been rejected on account of colour vision, the average sea service being eight years, eight men holding first mate certificates, the average sea service being fourteen years, and of four men holding master's certificates with an average of twenty-four years, but none had been involved in any disaster and he accepted the statistics as supporting his view that most colour blind people are safe.

On the other hand the personal experience of Captain Craig, of an anonymous gentleman and of a naval officer whose case was described by Nettleship, shows that danger exists. Such danger as does exist from colour blindness, would be reduced by eliminating, if it could be done, the green light which Lindo Ferguson criticized as not penetrating well, possibly due to increased absorption of green glass over that of red glass.

Regarding the visual acuteness necessary, Mackay, of Edinburgh, considers two diopters of hypermetropia a hindrance, while Freeland Fergus with an artificial myopia of three diopters could locate piers two and a half and five nautical miles away and he would accept as safe at sea anyone who could pick up a buoy over a mile away or a vessel three to four miles off, although he did not care to translate the empirical standard into terms of Snellen's tests. My only experience bearing on this point has been two cases of acute asthenopia in yachtsmen of low hypermetropia trying to pick up landmarks and buoys on a dark night; the fatigue was sufficient to require a total rest from any accommodative effort for some days.

I believe with Freeland Fergus that the "form sense" is essential and the advisability of an investigation of the light minimum and light difference senses which might be easily accomplished by such an apparatus as Percival's, of Newcastle. One has only to reflect on one's personal experience at sea to see the force of his arguments which in another sphere apply to the finding of one's golf ball, which too frequently becomes camouflaged against its background.

Quoting statistics compiled from the Board of Trade Wreck Abstracts of fourteen years of the collisions about

the coasts of the United Kingdom and British Possessions abroad and Table I. (Appendix B.) of the same evidence show of a yearly average of 1,092 collisions. One hundred and eighty were due to unknown causes and one hundred to bad look-out.

Of these some might quite well be explained by defective vision and it would be interesting to hear from Dr. Ferguson the answer put to him by the Committee as to whether there has been any difference in the incidence of accidents that might be attributed to defective eyesight before and after the institution of the Union Company's new standard of tests in New Zealand.

THE CAUSES OF BLINDNESS.

By James W. Barrett, K.B.E., C.B., C.M.G., M.D., M.S., F.R.C.S.,
Melbourne.

I have been acting as oculist to the Royal Victorian Institute to the Blind since 1901 and during that period the applicants for admission to the Institute have been referred to me before they have been enrolled. Somewhere about two hundred and fifty applicants have in this way passed through my hands. One hundred and ninety-two have been selected for analysis as the notes were sufficiently comprehensive to enable conclusions to be drawn with every degree of probability. It will be noted that the majority of the persons were children or adolescents since the Institution is educational in its objects. There are at present in the State of Victoria eleven hundred blind people. No doubt a number of these have become blind in late adult life, consequently it is more than probable that the causes of blindness as set out in my table would not agree with the causes of blindness for the whole of the eleven hundred. This caution should be born in mind because the result of my analysis may seem startling. The following table summarizes the result of my inquiry.

Disease	Number of Persons	Percentage of Total
Optic atrophy	47	24
Chorioiditis	21	11
Ophthalmia neonatorum	21	11
Corneal ulceration	15	8
Injury	14	7
Trachoma	13	6.7
Nystagmus	10	5.2
Cataract	9	5
Buphthalmos	9	5
Degeneration of vitreous	5	2.5
Shrunken globes	5	2.5
Kerato iritis	4	2
Retinitis pigmentosa	4	2
Doubtful	2	1
Interstitial keratitis	3	1.5
Cerebro-spinal meningitis	2	1
Glioma	2	1
Microthalmos	2	1
Pan-ophthalmitis	1	
Myopia	1	
Retinal haemorrhage	1	
Degeneration of the cornea	1	
All disease	192	—

The persons set down as suffering from nystagmus showed no lesion, but they had nystagmus and were practically blind.

In those with corneal ulceration it was impossible to assign the condition to any particular cause from the history given.

For convenience of reference the eye conditions have been classified as total blindness, almost total blindness and partial blindness. Total blindness meaning inability with both eyes to count fingers at a distance of two to three metres in any circumstances; almost total blindness means vision of less than 6/60 in any circumstance and partial blindness means vision of less than 6/36.

Optic Atrophy.

Of the forty-seven persons the oldest was forty years of age and the youngest five. The average age of forty-two was fifteen years and the ages of five were not definitely known. The blindness was partial in six persons, almost total in two and total in the remainder.

It must be remembered many of these persons were seen before a Wassermann test was practicable, but when it has been possible to obtain a Wassermann test, the weakness of the clinical history became obvious. For example, in the case of one child the blindness was attributed to meningitis, but the Wassermann test gave a partial positive result. Very few of the patients showed any other sign of syphilis and in the majority of the cases there was no explanation given of the atrophy. The list may include one or two cases of Leber's disease, but my impression is the great majority were syphilitic.

Chorioiditis.

Of these persons the oldest was forty-eight and the youngest six years of age. The average age was eighteen and the age of one was unknown. The blindness was total in eleven persons, almost total in seven and partial in three. In two of the patients the Wassermann test yielded a reaction.

Ophthalmia Neonatorum.

The youngest patient was five and a half years and the oldest twenty-five. The average age of nineteen was fourteen years. The serum of one of the patients gave a partially positive reaction to the Wassermann test. One child with corneal opacity in the right, shrunken globe in the left and partial blindness gave a partial reaction to the Wassermann test. Another child with nebulæ and partial blindness said to date from three days after birth gave the Wassermann reaction.

Corneal Ulceration.

The youngest was six and the oldest forty-eight. The average age of fourteen was nineteen years and the age of one was unknown. Of these eleven were totally blind and three were partially blind.

Injury.

The youngest was seven years of age, the oldest thirty-six. The average age of twelve was seventeen and the

ages of two were unknown. The blindness was total in thirteen persons and partial in one. In one the right eye was said to have been lost through an explosion, but the left eye was blind from trachoma. The misleading nature of the history given may be gauged from the following. A patient ascribed her condition to running scissors into the left eye which was removed after several operations. The right eye became affected one month after the accident. She came to see me with a shrunken globe, complaining of great pain. The Wassermann test, however, yielded a good partial result and the symptoms were relieved by anti-syphilitic treatment. Another person with total blindness attributed his condition to a boiler explosion. The cause was, however, extensive chorioiditis. Another patient informed me that the left eye had been excised to save the right and there was evidence of old iritis in the right, but the Wassermann test yielded a definite reaction.

Trachoma.

The youngest was eleven years of age and the oldest fifty-four. The average age of twelve was twenty-three and the age of one was unknown. The blindness was total in six persons, almost total in six and partial in one.

Nystagmus.

The youngest was seven and the oldest twenty-nine. The average age was twelve and a half. The blindness was total in four, almost total in four and partial in two. The serum of one person was tested and a Wassermann reaction was obtained.

Sympathetic Ophthalmia.

Under the heading of injury and corneal ulceration there may be seven cases of sympathetic ophthalmitis, but as the Wassermann reaction was not taken in these cases, I have hesitated to classify them as such. In another case assumed to be sympathetic ophthalmia the present condition is one of trachoma.

Cataract.

The youngest was eight and the oldest was nineteen years of age. The average age was ten, but probably a number of these persons were syphilitic and the cataract, although congenital, was secondary. They did not recover vision after successful extraction. In one patient the Wassermann test yielded a positive reaction. The blindness was total in three persons, almost total in two and partial in four.

Degeneration of the Vitreous.

The youngest was five years, the oldest fifty-six. The serum of one child aged five was examined and the Wassermann test yielded a partially positive response. The persons with shrunken globes were all children and the causes were unknown.

Kerato-Iritis.

The persons were young adults and the blindness total or almost total.

Retinitis Pigmentosa.

The average age was twenty-six. The blindness was total in three persons and partial in one.

Glioma.

There were two persons with double excision for glioma who had survived.

Conclusion.

On surveying the list it appears that in the majority of cases atrophy and chorioiditis were certainly due to syphilis, that the cases of *ophthalmia neonatorum* were due to gonorrhœa, that some of the cases of corneal ulceration were due to syphilis or gonorrhœa and that some percentage of the cases of nystagmus, cataract, buththalmos, degeneration of the vitreous and keratoiritis were due to syphilis and that the case of interstitial keratitis was definitely syphilitic. Wherever it was possible to test this hypothesis by the Wassermann test or by family history the result indicated syphilis as a cause. It is therefore conservative to attribute at least 50% of these cases of blindness to venereal disease and yet very few of the persons showed any external sign of venereal disease beyond the eye condition. It is for this reason that the statistics of the causes of blindness taken before the days of Wassermann test are so liable to mislead.

Dr. D. D. Paton read some statistics from the blind school in Western Australia. The period covered was from 1900. There were thirteen congenital defects including three instances of buphthalmos. Six persons were blind from trachoma. One was forty-one years of age and had been admitted in 1900, one was twenty years of age and had been admitted in 1908, one was ten years of age and had been admitted in 1912. The condition had been greatly neglected in this patient. One was eleven years of age and had been admitted in 1917, one was twenty-three years of age and had been admitted in 1919 and the sixth was forty years of age and had been admitted in 1923. Three persons had gonorrhœal ophthalmia. Their ages were ten, thirty-eight and thirty-three years respectively. Fifteen persons had become blind as a result of some accident. In two instances the injury was a war injury. Explosion was the cause of the injury in nearly every case. Twenty persons had optic atrophy. Six of them were under eleven years of age. In two instances the optic atrophy was secondary. There were two with Leber's atrophy, eight with perforating corneal ulcers and eight with corneal nebula, five with *phthisis bulbi*, six with retinitis, two with post-operative blindness, one with detached retina, three with sympathetic ophthalmia and six in whom the blindness was undefined. The total number of blind persons was ninety-eight.